A Study of
Coronary Angiographic profile
in Patients undergoing Permanent Pacemaker implantation

Dissertation submitted to
The Tamil Nadu Dr M.G.R Medical University, Chennai
in partial fulfillment of the requirements for the degree of
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Certificate

This is to certify that Dr V.Rajendran postgraduate student [2003-2006] in the Department of Cardiology, Government General Hospital Chennai & Madras Medical College, Chennai -03, has done this Dissertation on "A Study of Coronary Angiographic profile in Patients undergoing Permanent Pacemaker implantation" under my guidance and supervision in partial fulfillment of the regulations laid down by The Tamil Nadu Dr M.G.R Medical University, Chennai, for DM Cardiology – Branch II examination to be held in February 2006.

Dean

Madras Medical College

Government General Hospital

Chennai-03.

Prof. Dr.V. Jaganathan., MD.,DM,.

Prof and Head

Department of Cardiology

Government General Hospital &

Madras Medical College

Chennai-03.

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CONTENTS

INTRODUCTION

Symptomatic bradyarrhythmia occurs most often in aged patients. Most of these patients have multiple coronary risk factors and present with angina-like symptoms. The coexistence of CAD not only has major effects on their prognosis but also influences the long-term care. This study was designed to evaluate the incidence of coexistent CAD in patients with symptomatic bradyarrhythmias and its relationship to conventional coronary risk factors in our population.

From May 2004 to June 2005, we prospectively studied all consecutive patients admitted to our institution for symptomatic bradyarrhythmias requiring permanent pacemaker implantation. Coronary angiographies were performed non-selectively at the same session of pacemaker implantation. Based on the presence or absence of CAD, patients were divided into two groups for analysis. Multivariate logistic regression analysis was performed to determine independent predictors of CAD including sex, age, diabetes mellitus (DM), hypertension, hypercholesterolemia, and smoking. The odds-ratio (OR) was determined.

In our patients with symptomatic bradyarrhythmias requiring permanent cardiac pacing, the incidence of CAD was 31 % as determined by coronary angiography (CAG). Male sex, hypertension, hypercholesterolemia and diabetes mellitus were the significant predictors for CAD in these patients. The Av nodal artery was commonly involved in patients with coexistent CAD and symptomatic bradyarrhythmias due to AV block.

Overview

Bradycardia is most commonly secondary to sinus nodal dysfunction. Bradyarrhythmias secondary to AV block at the level of the AV node or distal to the bundle of His also account for a large number of patients who have bradyarrhythmias.

Sinus nodal dysfunction and atrioventricular (AV) block account for the majority of significant bradyarrhythmias. In addition to structural abnormalities, drug effects and autonomic influences can cause sinus nodal dysfunction. Acquired AV block is most commonly caused by idiopathic fibrosis, acute myocardial infarction, or drug effects. Patients with asymptomatic sinus bradycardia or sinus pauses have a good prognosis and do not require treatment. On the other hand, those with tachycardia-bradycardia syndrome have a much worse prognosis, because of their risk of thromboembolic complications. Therefore, the aim of therapy is prevention of atrial fibrillation. Atrial pacing and anticoagulation can greatly reduce the incidence of stroke in this high-risk group. Once appropriate pacing has been established for AV block, the prognosis is primarily dependent on the extent of the associated heart disease.

The availability of pacing especially dual-chamber and rate-responsive pacing has greatly improved the lifestyle and prognosis of symptomatic patients.

Anatomy and Blood supply of conducting system[12]

SA Node

In humans, the sinus node is a spindle shaped structure composed of a fibrous tissue matrix with closely packed cells. It is 10 mm to 20 mm long and 2 to 3 mm wide, and thick, tending to narrow caudally toward the inferior vena cava. It lies less than 1mm from the epicardial surface, laterally in the right atrial sulcus terminalis at the junction of the superior vena cava and right atrium.

Blood supply of SA node

The artery supplying the sinus node branches from the right coronary artery in 55 to 60 percent of the time, the left circumflex coronary artery in 40 to 45 percent of time and approaches the node from a clockwise or counterclockwise direction around the superior vena caval-right atrial junction.

AV Node and Internodal Connection

Internodal and intraatrial conduction occurs through the presence of three interatrial pathways. These groups of internodal tissue are best referred to as internodal atrial myocardium, not tracts.

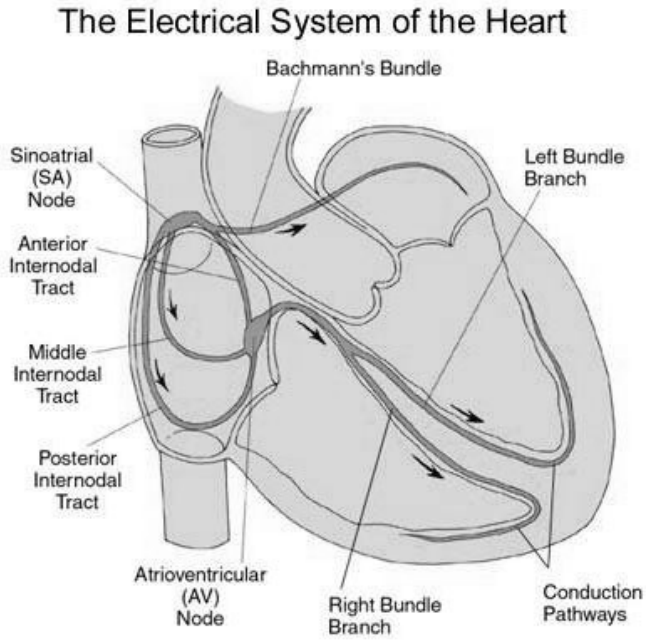
The normal AV junctional area can be divided into three distinct regions;

1. Transitional cell zone .
    - They differ from atrial myocardium and connect the the latter with the

compact portion of the AV node.

2. The Atrioventricular node
    - The compact portion of the Av node is a superficial structure. The AV node lies beneath the right atrial endocardium at the apex of the triangle of Koch, which is formed by the tendon of Todaro and the septal leaflet of the tricuspid valve. This positions the AV node directly above the insertion of the septal leaflet of the tricuspid valve and anterior to the ostium of the coronary sinus
3. Penetrating part of the AV Bundle.
    - At its distal end, the compact portion of the AV node enters the central fibrous body, becoming the penetrating portion, or bundle of His.

The main function of AV node is modulation of atrial impulse transmission to ventricles , thereby coordinating atria and ventricular contractions.



Blood Supply

of AV node

The blood supply to the AV node is via the AV nodal artery, a branch of the right coronary artery in 90% of hearts that originates at the posterior intersection of the AV and interventricular grooves, with the remaining 10% arising from the circumflex artery

His-Purkinje System[12]

The cells of the proximal bundle of His resemble those of the compact AV node, whereas the distal cells are similar to the cells in the proximal bundle branches. The bundle of His is contained within the connective tissue of the central fibrous body and membranous septum. It courses in the base of the membranous septum or along the left side of the crest of the interventricular septum in most cases.

The right bundle branch (RBB) extends unbranched from the bundle of His, traveling down the right side of the interventricular septum to the apex of the right ventricle, and the base of the anterior papillary muscle. Discrete bifascicular subdivisions with anterosuperior and posteroinferior branches of the LBB are not the norm. Despite this anatomic variability, the concept of the left anterior and posterior fascicular blocks remains clinically useful. The bundle branches end in Purkinje’s fibers, forming a network over the surface of the endocardium of the ventricles.

Blood supply of His Bundle

The bundle of His has a dual blood supply from branches of the anterior and posterior descending coronary arteries. Dual blood supply from the left and right coronary arteries is the usual case for the bundle branches .

Sinus Nodal Dysfunction

Historical Perspective

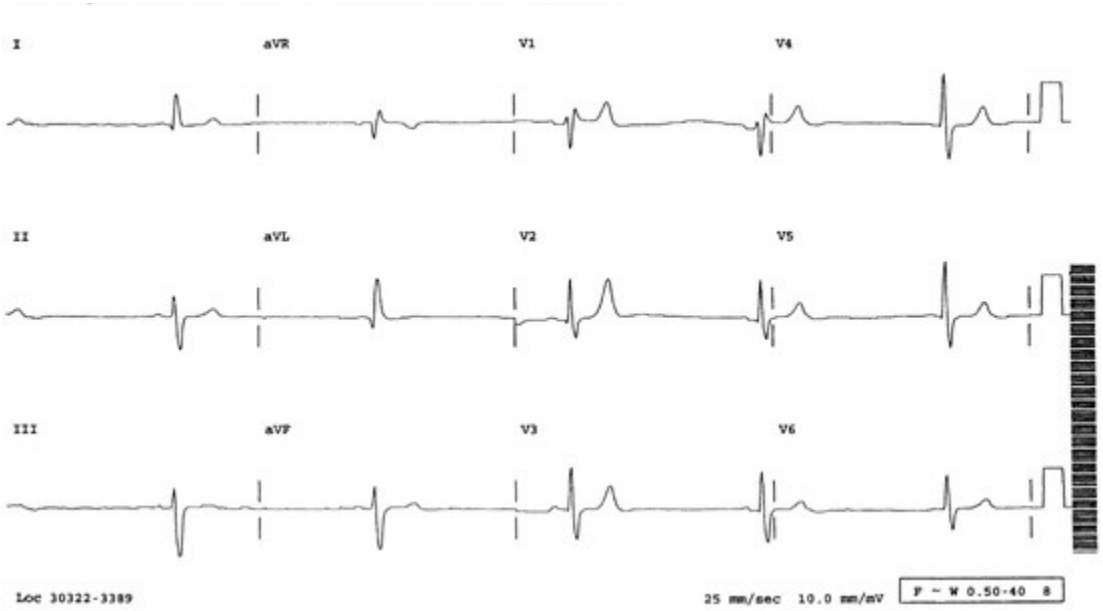
Rhythm disturbances resulting from sinus nodal dysfunction include sinus bradycardia and pauses, as well as atrial tachyarrhythmias alternating with bradyarrhythmias. As early as 1909, episodes of atrial standstill of 4 to 8 seconds in duration were documented by Laslett in a patient with syncope, using combined jugular venous pulse and radial artery tracings [1]. This report was followed by Mackenzie's recordings of digitalis-induced atrial standstill [2]. These early reports identified the association of atrial standstill with syncope and the possible adverse effects of drugs on the patient with sinus nodal dysfunction. However, the concept of sinus nodal dysfunction as a clinical syndrome did not appear until Short described alternating bradycardia and tachycardia in patients with syncope [3]. Some clarification of this association between bradycardia and tachycardia was provided by Katz and Pick in 1956 in their description of overdrive suppression of the sinus node, resulting sinus standstill, and the danger of asystole when subsidiary pacemakers fail [4].

In 1967, Lown coined the phrase *sick sinus syndrome* for describing chaotic atrial activity combined with bradycardia postcardioversion 5. This term was further popularized by Ferrer, who defined the spectrum of arrhythmias involved 6,7. The clinical entity of sick sinus syndrome became widely accepted as a frequent and important management problem. Although *sick sinus syndrome* is still used, the terms *sinus nodal dysfunction*, *sinoatrial disease*, and *sinoatrial dysfunction* are more commonly used to describe this syndrome.

Electrocardiographic Features of Sinus Nodal Dysfunction

Inappropriate Sinus Bradycardia

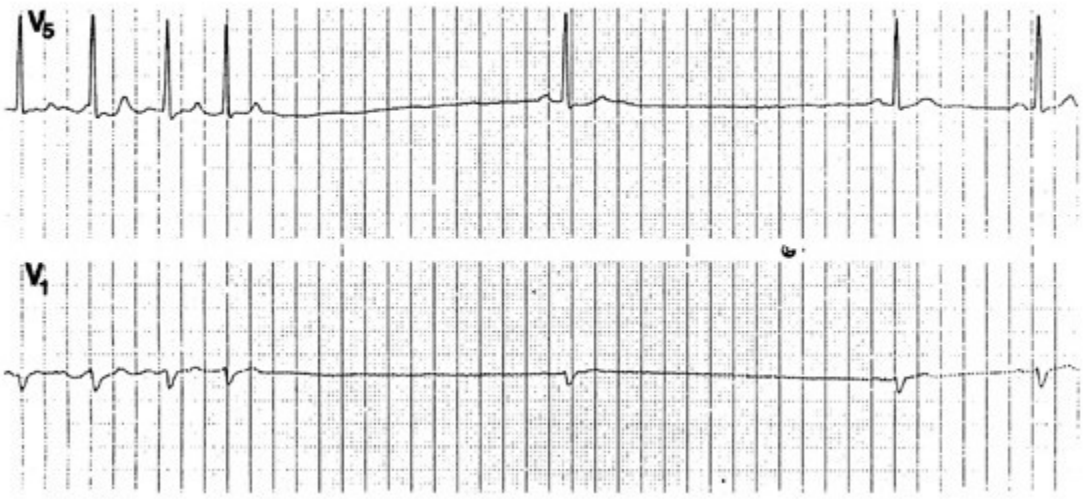
Sinus bradycardia (sinus rate less than 60 beats/min) is considered inappropriate when it is persistent and does not increase appropriately with exercise.



Sinus Arrest

The terms *sinus arrest* and *sinus pause* are used interchangeably and are a result of the sinus node's principal pacemaker cells failing to fire. The pause is not an exact multiple of the preceding PP interval.

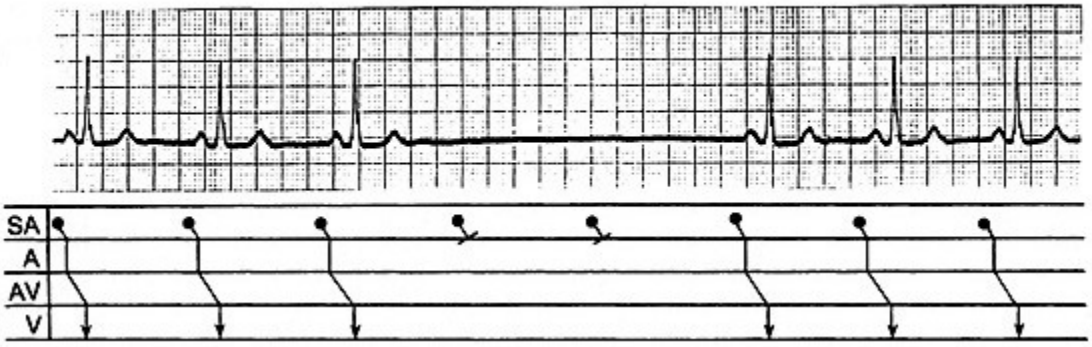
Pauses greater than 3 seconds are rare in normal individuals and may or may not be associated with symptoms, but are usually caused by sinus nodal dysfunction



Sinoatrial Exit Block

Like sinus arrest, sinoatrial exit block is characterized by a period of asystole without the expected P wave seen. In patients with sinus nodal dysfunction, an escape beat does not appear, but instead the sinus block is terminated by a sinus beat. In theory, sinoatrial exit

block can be distinguished from sinus arrest because the exit block pause is an exact multiple of the baseline PP interval



This rhythm strip is an example of sinoatrial exit block.

Sino atrial exit block is classified into three types, analogous to that of AV block. First-degree sinoatrial block is caused by abnormal prolongation of the sinoatrial conduction time. This conduction abnormality cannot be identified from surface electrography, but needs to be verified either by direct recording or indirect measurement of sinoatrial conduction time during an electrophysiologic study.

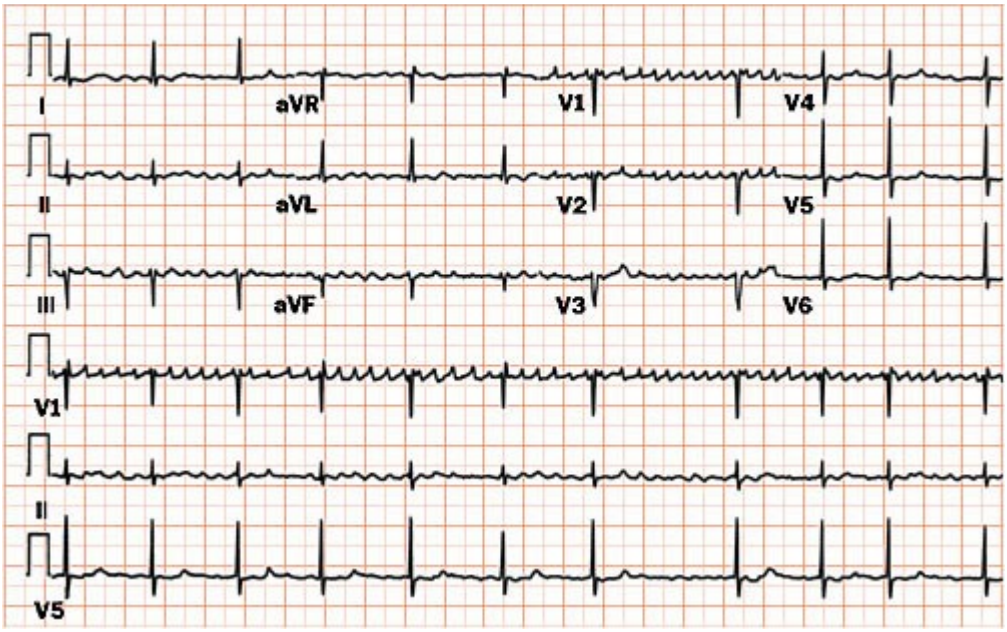
Second-degree sinoatrial exit block is marked by intermittent failure of the sinus impulse to exit the node. This is viewed as Wenckebach periodicity of the P wave on surface electrography. Progressive shortening of the PP interval preceding the dropped P wave is seen.

In higher grade, second-degree sinoatrial exit block, abrupt absence of one or more P waves results from failure of the atrial impulse to exit the sinus node. As noted previously, the atrial pause should be an exact multiple of the immediately preceding PP

interval. Third-degree or complete sinoatrial exit block is seen as a pause in the sinus rhythm and cannot be distinguished from prolonged sinus arrest

Chronic Atrial Fibrillation

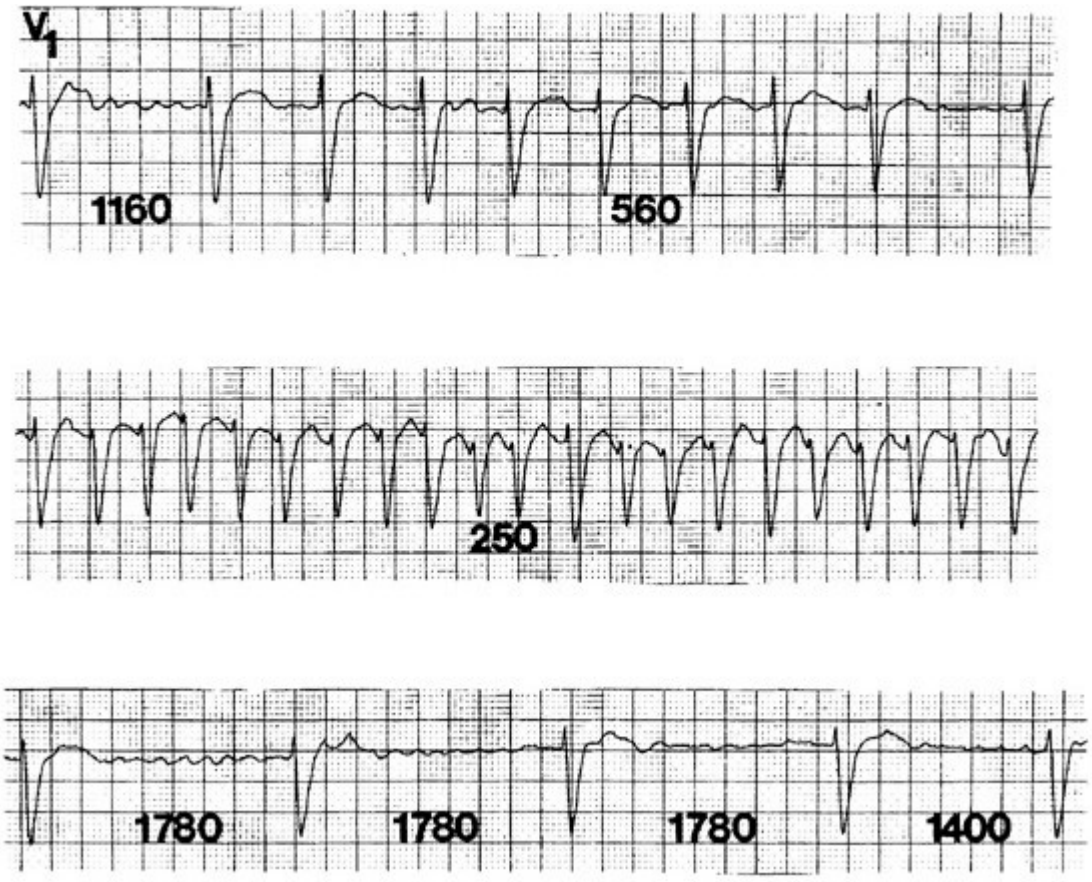
The presence of chronic atrial fibrillation in a patient with a slow ventricular response not secondary to drug therapy is a sign of sinus nodal dysfunction..



Atrial fibrillation. Narrow complex QRS with irregular RR intervals. Ventricular rate is controlled.

Tachycardia-Bradycardia Syndrome

Tachycardia-bradycardia syndrome refers to the presence of intermittent sinus or junctional bradycardia alternating with atrial tachycardia (usually paroxysmal atrial fibrillation) in the same patient. The highest incidence of syncope associated with sinus nodal dysfunction probably occurs in this group [3].



These three rhythm strips were obtained from the same patient at different times and illustrate the marked variability of heart rates in patients with tachycardia-bradycardia syndrome. The labeled cycle lengths are in milliseconds.

Pathophysiology

Idiopathic degenerative disease is probably the most common cause of intrinsic sinus nodal dysfunction. Pathologic studies have shown an increase in fibrous tissue in the area of the sinus node with age. Why some individuals develop sinus nodal dysfunction whereas others do not, is not easily explained by fibrous tissue replacement only. Coronary artery disease may be responsible for one-third of cases of sinus nodal dysfunction. This estimation is based on a study by Shaw et al. in which angiography was used to demonstrate the extent of coronary artery disease in the sinus nodal artery in patients with sinus nodal dysfunction. Transient slowing of the sinus rate, or sinus arrest, can complicate an acute myocardial infarction. This is usually seen with an acute inferior wall myocardial infarction, is caused by neural influences, and rarely persists.

Coronary artery disease may coexist with sick sinus syndrome in a significant number of patients, although it is not considered a major cause of the syndrome. It is unclear whether inflammation, sinus node ischemia, or local autonomic neural effects lead to the development of sick sinus syndrome in patients with myocardial infarction. Sinus node dysfunction usually is temporary when it follows an acute myocardial infarction. Uncommonly, chronic ischemia may cause fibrosis and lead to symptoms of sick sinus syndrome for months to years after myocardial infarction.

Diagnostic Techniques

Both noninvasive and invasive means of diagnosing sinus nodal dysfunction are available. Generally, the noninvasive methods of electrocardiographic (ECG) monitoring, exercise testing, and autonomic testing are used first. However, if symptoms are infrequent, invasive electrophysiologic testing may be pursued.

Management

Sinus nodal dysfunction is currently the most common reported diagnosis for pacemaker implantation. However, once the decision to pace is made, choosing the optimal pacemaker prescription is essential to decrease stroke risk and improve quality of life. As discussed previously, a significant number of patients with sinus nodal dysfunction have concomitant atrial fibrillation or develop new atrial tachyarrhythmias after being diagnosed with sinus nodal dysfunction. Atrial pacing has been shown to greatly decrease the incidence of atrial fibrillation and thromboembolism in this population, whereas those who are only ventricularly paced have not seen a similar benefit. For those patients with sinus nodal dysfunction who have normal AV conduction, a single-chamber atrial pacemaker is a reasonable consideration. Of course, single-chamber ventricular pacing is still appropriate for individuals with sinus nodal dysfunction and chronic atrial fibrillation.

Guidelines for implantation of pacemakers in patients with sinus nodal dysfunction have been established by a task force from the American College of Cardiology and American Heart Association

Indications for permanent pacing in sinus nodal dysfunction[a]

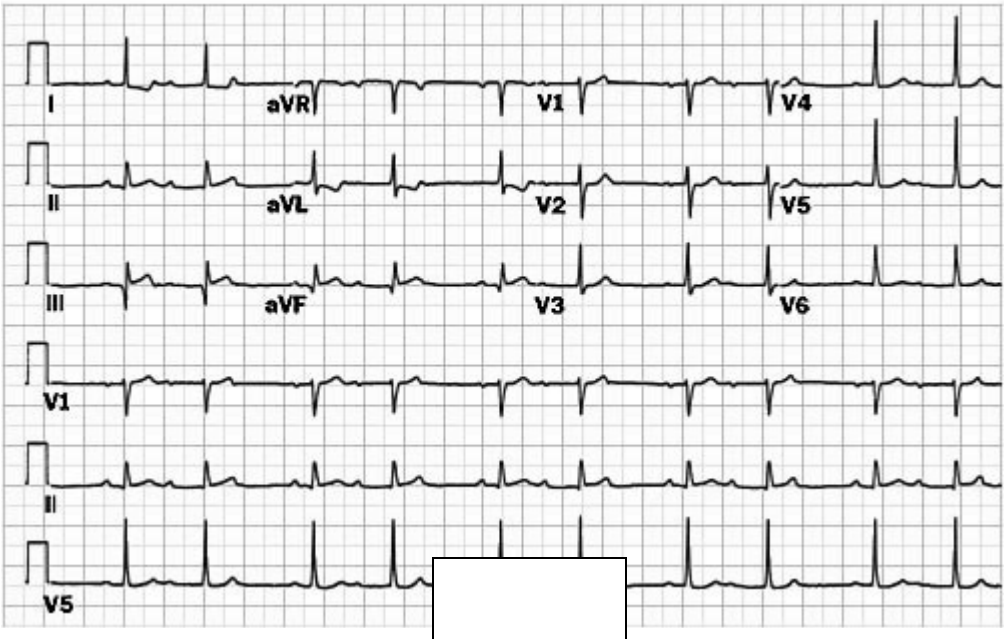
Class I	
Sinus nodal dysfunction documented in association with symptomatic bradycardia and caused by factors that are irreversible or caused by essential drug therapyClass II	No clear association between sinus nodal dysfunction (with heart rate <40 beats/min) and symptoms can be documented
Class III	Sinus nodal dysfunction with marked sinus bradycardia or pauses, but no associated symptoms

[a]Class I indications are conditions for which it is generally agreed that pacing is needed. Class II indications are conditions associated with varying opinions as to the necessity of pacing. Class III indications are conditions in which pacing is thought not to be indicated.

Atrioventricular Nodal Dysfunction

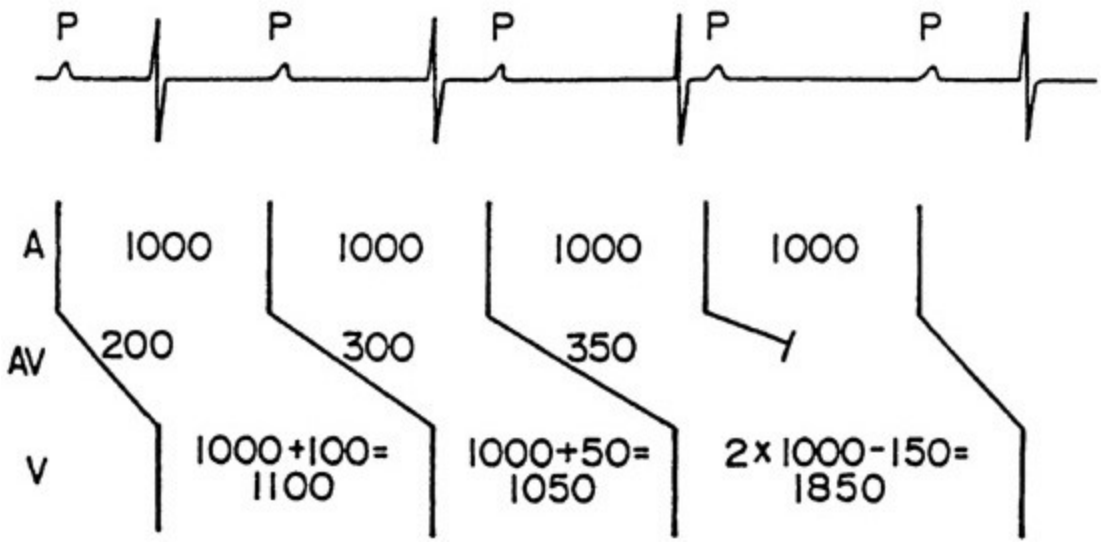
Historical Perspective

Adams (1827) and Stokes (1846) first described syncope associated with a slow heart rate. In 1906, by analyzing the jugular venous pulse waves, Wenckebach and Hay were able to distinguish two types of second-degree AV block. Once the ECG became available, Mobitz classified them as types I and II. With the development of intracardiac electrocardiology and bundle of His recordings in the late 1960s, knowledge of AV conduction was expanded. The ability to localize the site of the block to the AV node, bundle of His, or bundle branch fascicular system was facilitated. After the development of chronic transvenous ventricular pacing in the mid-1960s, the natural history of the patient with symptomatic heart block greatly changed, and mortality significantly decreased. Further advancements in pacing over the years—namely, dual-chamber and rate-responsive pacing—have improved the quality of life of these patients.



Acute inferior myocardial infarction complicated by Wenckebach block. Note inferior ST-segment elevation and Q waves as well as progressive prolongation of the PR interval with grouped beating.

First-Degree Atrioventricular Block First-degree AV block on the surface ECG is seen as a PR interval >0.20 seconds in adults or >0.18 seconds in children. Each P wave is followed by a QRS complex with a constant, prolonged interval. Although the conduction delay can be anywhere along the system, the PR prolongation is usually caused by delay within the AV node (87% when the QRS complex is narrow). On the bundle of His electrogram, this would be seen as an AH interval >130 ms with a normal HV interval . In cases in which first-degree AV block is seen in the presence of a bundle branch block, a bundle of His electrogram is necessary to localize the site of block . Infranodal conduction delay is present in 45% of these cases. A combination of delay within the AV node and in the His-Purkinje system must also be considered. In certain cases of congenital structural heart disease, such as Ebstein's anomaly of the tricuspid valve or endocardial cushion defects, intraatrial conduction delay can cause first-degree AV block. In addition, intra-Hisian conduction delay can cause first-degree AV block. On the bundle of His electrogram, a split His potential can be seen, resulting in a prolonged His potential, HV, and PR intervals. Dual AV nodal physiology can produce transient, abrupt, or alternating first-degree block caused by block in the fast AV nodal pathway (which is normally used), with conduction down the slow pathway instead. The change in the PR interval seen on the surface electrogram corresponds with a jump in the AH interval viewed on the bundle of His electrogram



Schematic of typical Wenckebach periodicity. The top panel shows a schematic electrocardiogram of a 4:3 Wenckebach cycle with prolonging PR intervals until the fourth P wave fails to conduct to the ventricle. On the bottom is a diagram illustrating the atrial (A), atrioventricular (AV) junctional, and ventricular (V) levels. Note that there is increasing conduction delay with each PR interval, but the amount of increase in conduction delay with each subsequent PR interval decreases. Therefore, the longest RR interval at the right of the figure is less than twice as long as the shortest RR interval immediately preceding it (see text for discussion). (From Miles MM, Klein LS. Sinus nodal dysfunction and atrioventricular conduction disturbances. In: Naccarelli GV, ed. *Cardiac arrhythmias: a practical approach*. Mount Kisco, NY: Futura, 1991:261, with permission.)

Second-Degree Atrioventricular Block

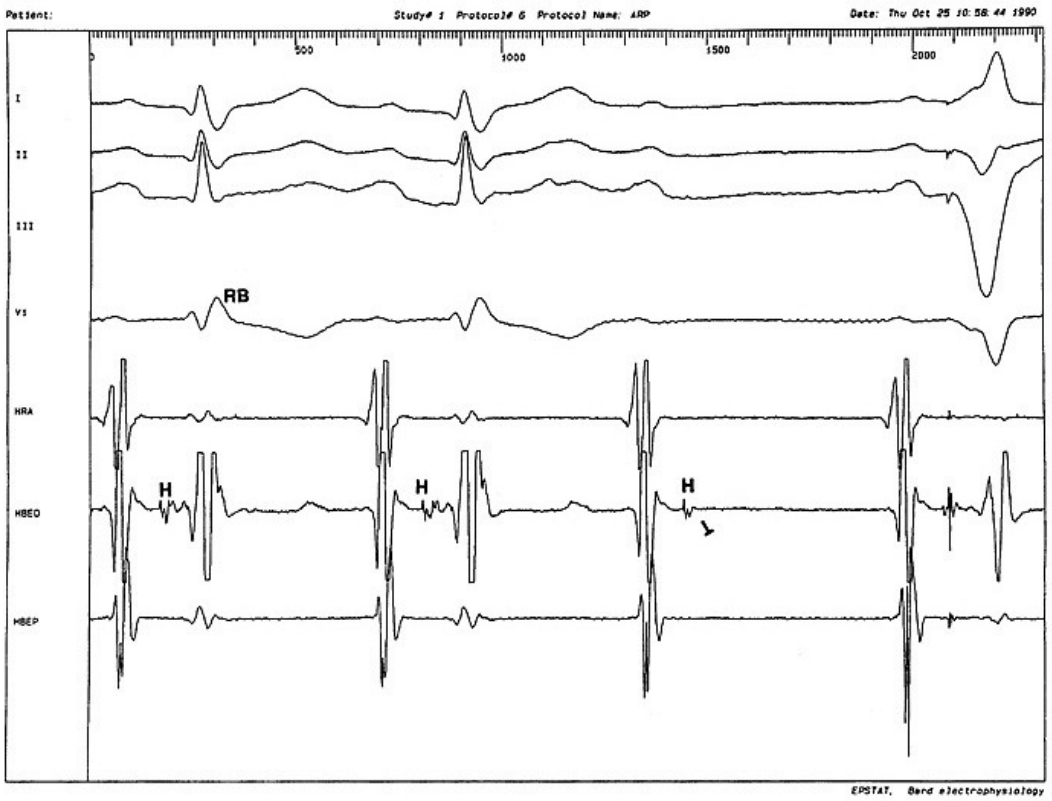
Type I

Type I second-degree AV block, or Wenckebach block, features on the surface electrogram progressive prolongation of the PR interval before failure of an atrial impulse to be conducted to the ventricles. The PR interval immediately postblock returns to its baseline interval, and the sequence begins again. Features of typical Wenckebach periodicity include the following

- Progressive lengthening of the PR interval throughout the Wenckebach cycle
- Lengthening of the RR interval occurring at progressively decreasing increments, resulting in progressive shortening of the RR intervals
- A pause including the non-conducted P wave that is less than the sum of any two consecutively conducted beats
- Shortening of the PR interval postblock, compared with the PR interval just preceding the blocked cycle

Less than 50% of type I AV block cases follow this typical pattern. Atypical patterns are more likely found with longer Wenckebach periods, >6:5. Differentiating atypical from typical patterns, however, is of little clinical significance.

Wenckebach block is almost always within the AV node when a narrow QRS complex is present. Intra-Hisian block is the rare exception. When type I block is seen with a bundle branch block, the block is still more likely to be in the AV node, but it could also be localized below the bundle of His. A bundle of His electrogram would be needed to accurately identify the level of block. Wenckebach block in the AV node is characterized by progressive prolongation of the AH interval, until an atrial deflection is not followed by a bundle of His or ventricular deflection. In type I block secondary to block below the bundle of His, progressive prolongation of the HV interval is followed by an H deflection without an associated ventricular depolarization.



The surface leads I, II, III, and V_1 show first-degree atrioventricular block and right bundle branch block (RB). The third P wave is not followed by a QRS complex. The intracardiac tracings [HRA, high right atrial recording; proximal (HBEP) and distal bundle of His electrograms (HBED)] reveal the diagnosis of block within the bundle of His. The His deflection (H) is fractionated. The third beat shows only the first half of the His, and no ventricular electrogram follows.

IMAGE

Type II

Type II, or Mobitz II, second-degree AV block is characterized on the surface electrogram by a constant PR interval, followed by sudden failure of a P wave to be conducted to the ventricles. The PP intervals remain constant, and the pause including the blocked P wave equals two PP intervals. Therefore, Mobitz II block should not be confused with a nonconducted premature atrial complex. Mobitz II block is usually associated with

bundle branch block or bifascicular block. In a majority of these cases, the site of block is within or below the bundle of His. When presumed Mobitz II block is seen in conjunction with a narrow QRS complex, Mobitz I with only minimal PR variation should be suspected. Only rarely is Mobitz II found with a narrow QRS complex and is caused by intra-Hisian block. The bundle of His electrogram is useful in verifying the site of the Mobitz II block. The blocked cycle features atrial and bundle of His deflections without a ventricular depolarization. The conducted beats usually show evidence of infranodal conduction system disease, with a prolonged HV interval, or even a split bundle of His potential.

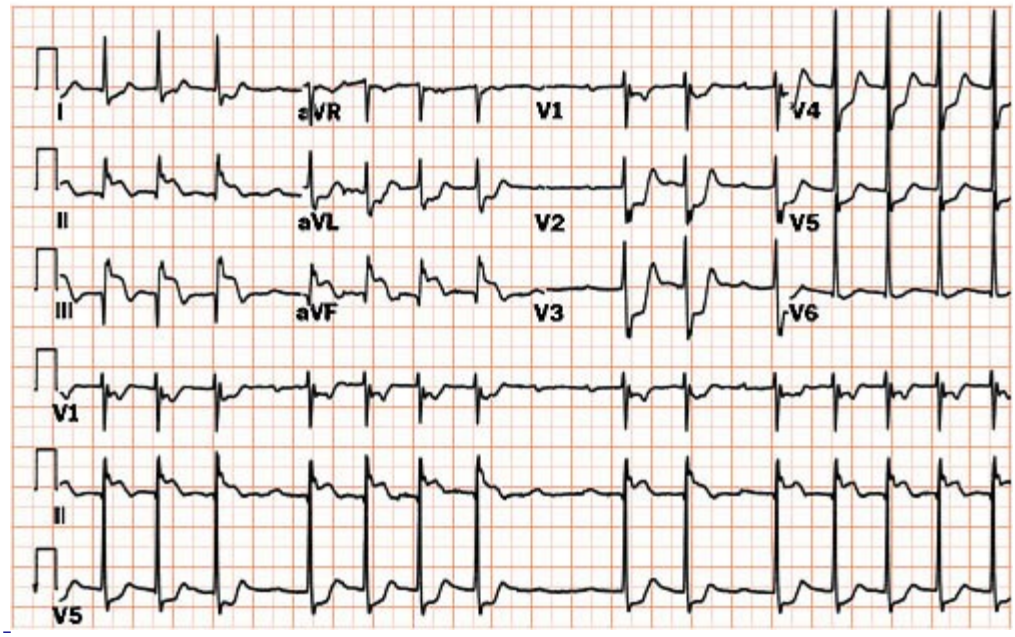
2:1 Atrioventricular Block

Fixed 2:1 AV block poses a diagnostic dilemma because it is usually impossible to classify as type I or II block by a surface electrogram alone. A narrow QRS complex and recently seen Wenckebach block is highly suggestive of block at the AV nodal level. A 2:1 block associated with a wide QRS complex is likely infranodal, but it could still be at the level of the AV node. A definitive diagnosis can only be made with an intracardiac recording at the bundle of His region.

Nonconduction of two or more consecutive P waves when AV synchrony is otherwise maintained is sometimes termed *high-degree AV block*. The level of block can be at the AV node or the His-Purkinje system. When high-degree AV block is caused by block in the AV node, QRS complexes of the conducted beats are usually narrow. Wenckebach periodicity is also seen, and atropine administration produces 1:1 conduction. Features pointing toward block in the His-Purkinje system are conducted beats with bundle branch block and no improvement in block with atropine. Bundle of

His recordings are sometimes needed to confirm the site of block.

Simultaneous surface tracings of leads V_1 and V_5 showing fixed 2:1 atrioventricular block and narrow QRS complexes. A definite diagnosis of either type I or II atrioventricular block cannot be made using this tracing alone.



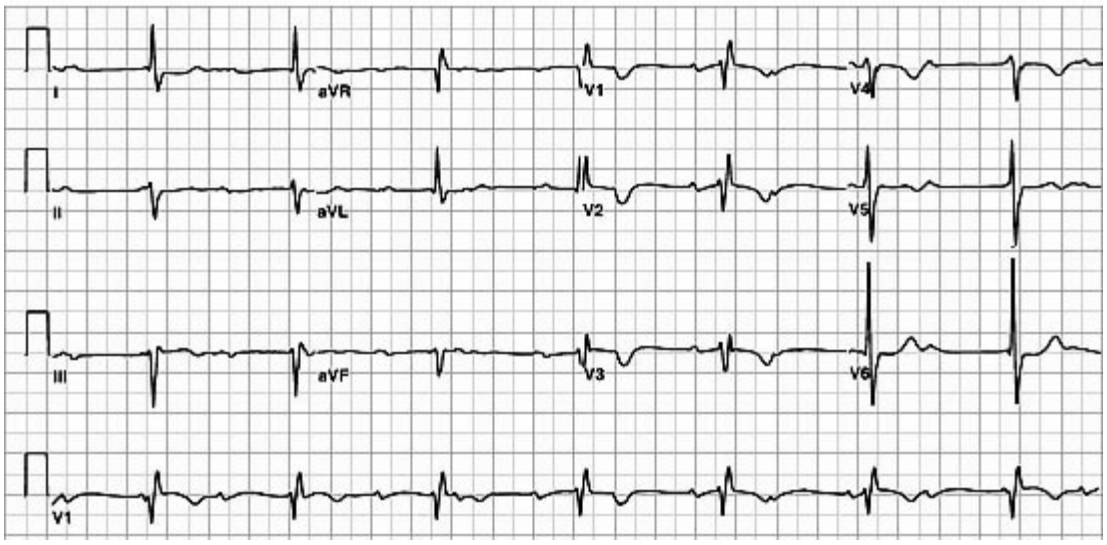
Acute inferior myocardial infarction with Mobitz II atrioventricular block. Note ST-segment elevation in the inferior leads as well as intermittently dropped QRS complexes.

IMAGE

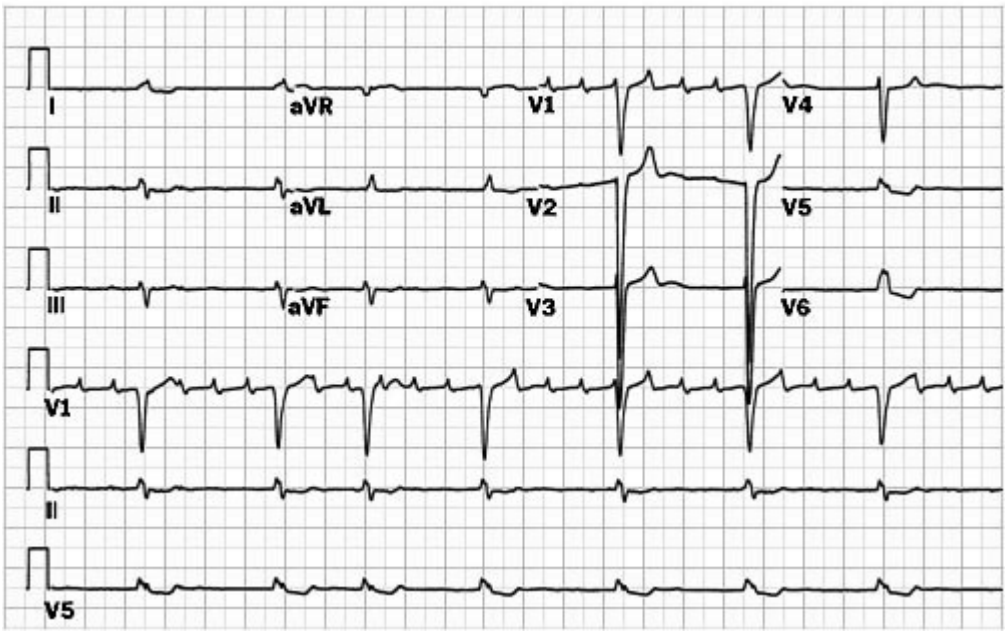
Third-Degree Atrioventricular Block

Third-degree, or complete, AV block is seen on the surface electrogram as completely dissociated P waves and QRS complexes, each firing at their own pacemaker rate. The atrial impulse is never conducted to the ventricles, but different levels of block are possible. The level of block determines the QRS morphology along with the site and rate of the escape rhythm. Congenital complete heart block is characterized by a narrow QRS complex with an escape rate between 40 and 60 beats/min, which tends to increase

with exercise or atropine. This is consistent with block within the AV node. Acquired complete heart block is usually associated with block in the His-Purkinje system, resulting in a wide QRS complex with an escape rate between 20 and 40 beats/min. The intracardiac electrogram shows bundle of His deflections consistently following the atrial electrograms, but the ventricular depolarization is completely dissociated from these. Block below the bundle of His is thus demonstrated. In contrast, complete heart block at the AV nodal level is seen on the intracardiac tracings as bundle of His potentials consistently preceding each ventricular depolarization. The atrial electrograms are dissociated from the HV complexes. The sinus rate is faster than the ventricular rate in patients with complete heart block. Data collected from patients with congenital complete heart block have showed the atrial rate to usually be age appropriate. It is important to note that complete antegrade AV block does not always predict retrograde (VA) conduction. Retrograde conduction may be intact in an individual with complete antegrade AV block



An 83-year-old patient with complete heart block, junctional escape rhythm, left anterior hemiblock, and complete right bundle branch block.



High-grade atrioventricular block. Note marked discrepancy between atrial and ventricular heart rates.

Atrioventricular Dissociation[12]

AV dissociation is a secondary phenomenon that results from a primary conduction disorder. It is characterized by the atria and ventricles depolarizing independent of each other. By definition, there is no retrograde conduction from the ventricles to the atria. AV dissociation may occur because of slowing or a failure of impulse formation or conduction from the sinus node. In this situation, the atrial rate may become slower than a subsidiary escape focus from the AV junction or ventricle. Thus, a competing AV junctional or idioventricular rhythm may develop. AV dissociation may occur if there is an increase in the automaticity of a subsidiary pacemaker, such as accelerated junctional rhythm or ventricular tachycardia.

AV dissociation may be complete or incomplete. In complete AV dissociation, both the atrial and ventricular rates remain constant, and, therefore, the PR interval varies, with none of the atrial complexes conducted to the ventricles. In incomplete AV dissociation (interference dissociation), ventricular capture beats occur because some of the atrial impulses arrive at the AV junction when the AV junction is no longer refractory. This phenomenon is common in advanced AV block with periodic capture beats.

Pathophysiology[12]

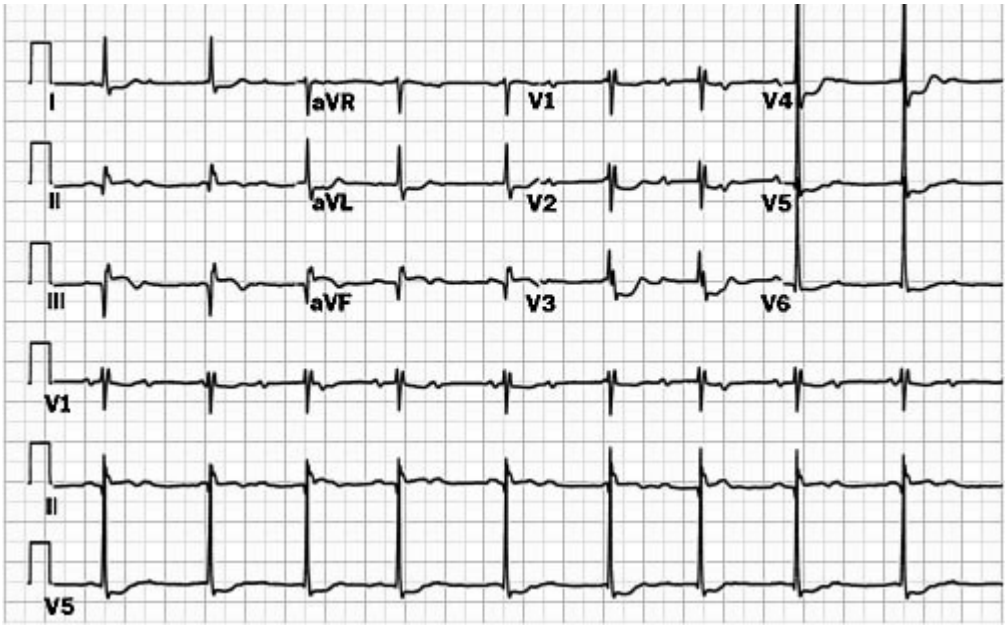
Acute myocardial infarction is associated with varying degrees of AV block and is the most common cause of acquired complete AV block. Second- and third-degree AV blocks occur in up to 30% of patients presenting with acute myocardial infarction.

Abnormalities in AV nodal conduction are seen in 20% of patients hospitalized for acute inferior myocardial infarction, with the onset of block falling in a bimodal distribution. Eleven percent of those presenting in the first hour of symptoms are found to have second- or third-degree AV block. In contrast, the incidence of heart block is low in the second hour of symptoms. The majority of conduction abnormalities occur between 2 and 72 hours. Because of the short duration of the early conduction abnormalities and their favorable response to atropine, an increase in vagal tone associated with acute inferior myocardial infarction is the probably etiology of this early phenomenon. Type I AV block occurring later in the course of an acute inferior myocardial infarction is less responsive to atropine and probably is associated with reversible ischemia of the AV node or the release of adenosine during acute infarction. In this setting, type I AV block rarely progresses to more advanced block and commonly resolves within 2 to 3 days of onset.

Type II AV block occurs in only 1% of patients with acute myocardial infarction, but it has a worse prognosis than type I block. It is associated with bundle branch infarction during an acute anterior myocardial infarction and frequently progresses to complete heart block.

Complete heart block can occur with either anterior or inferior acute myocardial infarction. The site of the block in inferior myocardial infarction is usually at the level of the AV node, resulting in a junctional escape rhythm with a rate of 50 to 60 beats/min and narrow QRS complex. The abnormality tends to be reversed with vagolytic drugs or exercise and usually resolves in several days. Complete heart block in the setting of acute anterior myocardial infarction is usually associated with infarction of the bundle

branches. The escape rhythm is approximately 40 beats/min, with a wide QRS complex originating from the bundle branch and Purkinje's system. It is less likely to be reversible. In general, patients who develop either transient or irreversible AV block are older and have a larger area of damage associated with their acute myocardial infarction. Other markers seen in this group include high levels of cardiac enzymes, bundle branch block, right ventricular infarction, or left ventricular failure.



Complicated acute inferior myocardial infarction. Note inferior ST-segment elevation as well as atrioventricular dissociation secondary to complete heart block.

In addition to acute myocardial infarctions, chronic ischemic heart disease can result in persistent AV block. Transient AV block can occur not only during angina pectoris, but during episodes of Prinzmetal's variant angina.

The most common cause of acquired conduction system disease is progressive idiopathic fibrosis. Lev's disease, is a result of proximal bundle branch fibrosis. It is postulated as a hastening of the aging process by hypertension and arteriosclerosis of the blood vessels supplying the conduction system. Lenègre's disease is a degenerative process occurring in a younger population and involving the more distal portions of the bundle branches.

Calcification of the aortic or mitral valve annulus can extend to the nearby conduction system and produce AV block. The incidence is more frequent with aortic than mitral stenosis. AV block can also result from a stenotic bicuspid aortic valve. Other causes of AV block include infiltrative cardiomyopathies such as amyloidosis, sarcoidosis, and hemochromatosis, as well as the collagen vascular diseases of scleroderma, rheumatoid arthritis, Reiter's syndrome, systemic lupus erythematosus, ankylosing spondylitis, and polymyositis.

Symptoms[12]

Individuals with first-degree AV block are asymptomatic. Symptoms of dizziness or syncope usually occur with acquired high-grade or complete AV block. With time, the majority of these patients experience a Stokes-Adams attack. Other symptoms can occur as a result of low cardiac output, including fatigue, congestive heart failure, dyspnea on exertion, angina, or even mental status changes.

Most children and adolescents with congenital complete heart block are asymptomatic, but some go on to develop symptoms later as adults.

Because the prognosis and the treatment differ in AV block depending on whether block is within the AV node or infranodal, determining the site of block is important. In many cases, this can be done noninvasively. As described previously, the QRS duration, PR intervals, and ventricular rate on the surface electrogram can provide important clues in localizing the level of block.

Management

Pacing is now the mainstay of treatment for symptomatic heart block. Temporary pacing is sometimes required in patients with acute myocardial infarction (anterior more often than inferior wall). Patients with asymptomatic first-degree or type I second-degree block do not require pacing. However, patients with type II second-degree or complete heart block should be temporarily paced, even if asymptomatic. If type II second-degree block or complete AV block persists once out of the periinfarct period, permanent pacing is indicated.

Even if the type II or third-degree block was transient but associated with bundle branch block, studies suggest permanent pacing of the postmyocardial infarction patient improves long-term survival. In the setting of myocardial infarction, the criteria for permanent pacing depends less on the presence of symptoms.

Indications for permanent pacing post–myocardial infarction[a]

Class I
Persistent high-grade or complete AV block with bilateral bundle branch block (infranodal)
Transient high-grade AV block associated with bundle branch block
Persistent, symptomatic second- or third-degree AV block
Class IIa
None
Class IIb
Persistent high-grade block at the AV nodal level
Class III
Transient AV block without intraventricular conduction defect
Transient AV block with isolated left anterior (or posterior) fascicular block
New left anterior (or posterior) fascicular block without AV block
First-degree AV block with new bundle branch block

AV, atrioventricular.

[a]Class I indications are conditions for which it is generally agreed that pacing is needed. Class II indications are conditions associated with varying opinions as to the necessity of pacing. The efficacy of pacing is less well established for class IIb than for class IIa. Class III indications are conditions in which pacing is thought not to be indicated.

PACEMAKERS[12]

The selection of the pacing mode should be based on the immediate needs of the patient and the potential long-term benefit of specific pacing modes. When determining the most appropriate pacing mode for a patient, the clinician should consider the patient's activity level, need for atrioventricular (AV) synchrony, need for chronotropic support, and associated medical conditions. When choosing a pacemaker, remember that rate-adaptive pacing technology allows, for most patients, restoration of a near normal chronotropic response Worldwide, VVI pacing is the most commonly used pacing mode. Although VVI pacing protects the patient from lethal bradycardias, it has significant limitations in that it does not restore or maintain AV synchrony or provide rate responsiveness in patients with chronotropic incompetence.

Ventricular-inhibited (VVI) pacing incorporates sensing on the ventricular channel, and pacemaker output is inhibited by a sensed ventricular event). VVI pacemakers are refractory for a period after a paced or sensed ventricular event, the *ventricular refractory period*. Any ventricular event occurring within this period is not sensed and does not reset the ventricular timer

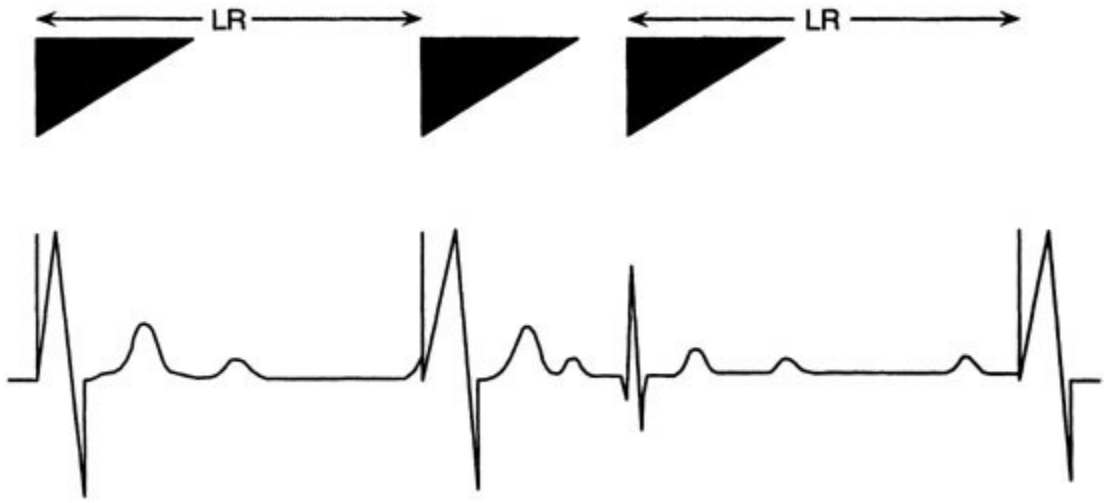
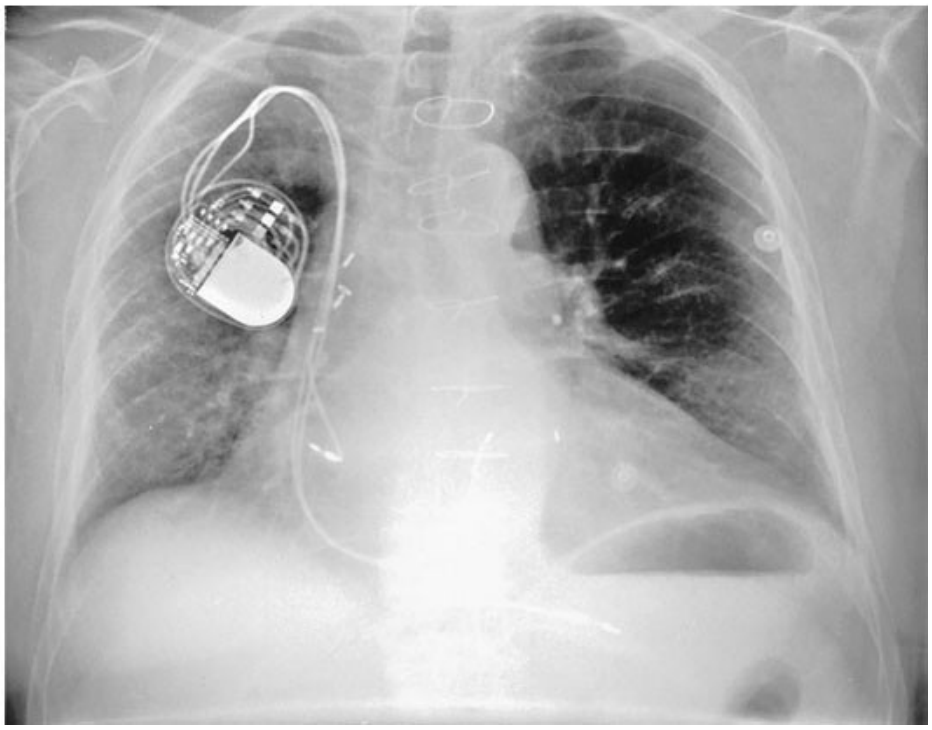


Figure 74.3 The VVI timing cycle consists of a defined lower rate (LR) limit and a ventricular refractory period (*black triangle*). When the LR limit timer is complete, a pacing artifact is delivered in the absence of a sensed intrinsic ventricular event. If an intrinsic QRS occurs, the LR

limit timer is started from that point. A ventricular refractory period begins with any sensed or paced ventricular activity. (From Hayes DL, Levine PA. Pacemaker timing cycles. In: Ellenbogen KA, ed. *Cardiac pacing*. Boston: Blackwell Scientific, 1992:263–308, with permission.)

The utilization and sophistication of permanent pacemakers have increased steadily since the first pacemaker implantation in 1958. Initially, pacemakers were indicated only for the prevention of Stokes-Adams attacks, but the indications have broadened as the technology has advanced. According to the estimates of four manufacturers, there were 152,909 primary pacemaker implantations (571 per million population) in 1997 and 37,946 pacemaker pulse generator replacements. Despite the continued sophistication and, at times, complexities of permanent pacemakers, they have become such a mainstay of therapy that it is imperative for clinical cardiologists to have a working knowledge of these devices.

A three-letter code describing the basic function of the various pacing systems was first proposed in 1974 by a combined task force from the American College of Cardiology and the American Heart Association (ACC/AHA) .



Postero-anterior **(A)** and lateral **(B)** chest radiographs demonstrating a ventricular lead that courses posteriorly in the coronary sinus and into a cardiac vein, probably a tributary of the posterior cardiac vein.

Review of Literature

Conduction disturbances of the heart may affect the sinoatrial and AV nodes and the intraventricular conduction system. The cause of severe cardiac conduction disturbances is often uncertain. Only few studies are available to find a common pathological and anatomic basis for conduction disturbances and the extent of coronary atherosclerotic disease that might be responsible for the conduction disturbances in patients with permanent pacemakers who underwent coronary arteriography The results of several small studies have been inconclusive.[1] [2] [3] [4] . In many patients, the cause is not known.

The relationship between conduction disturbances and the underlying pathology has been studied at autopsy, but these patients do not represent the general population, and many of the studies were conducted more than 20 years ago. Since then, the natural history of conduction disturbances has changed, longevity has improved, the spectrum of cardiac diseases has altered, and the treatment of conduction disturbances has been revolutionized by the use of pacemakers.

The association between conduction disturbances and atherosclerotic coronary disease has been investigated in only a few small studies in the early 1970s.[1] [2] [3] Coronary angiography performed in some series did not reveal specific coronary arterial lesions, and the anatomy of the branches supplying the conduction system was not studied.

Hamby et al[4] examined 42 patients with ECG conduction disturbances and symptomatic coronary artery disease. Although most patients had a significant lesion in the LAD coronary artery, there was no correlation with specific lesions. None of the patients in that study needed a permanent pacemaker.

Ruben-stein, *et al* found that 20 of their 56 patients with sick sinus syndromes had coronary artery disease based on either historical or electrocardiographic evidence;[1] Shaw, *et al* reported that 12 of their 25 patients with sick sinus syndromes had abnormal postmortem CAG.[2] Jordan, *et al* found significant sinoatrial conduction time (SACT) prolongation in patients with sinus nodal artery insufficiency and suggested a pathogenetic role of CAD in the development of sinus nodal dysfunction.[9]

However, Shaw, *et al* reported only 4 of 25 patients with chronic sinoatrial disorder had angiographic evidence of 75% stenosis obstructing the si nus nodal artery.

The Incidence of Coronary Artery Disease in Patients with Symptomatic Bradyarrhythmias

Hsueh, et al

(Jpn Heart J 2001; 42: 417-423)

This was the first human study concerning the incidence of CAD in Asian patients receiving permanent pacemaker implantation for symptomatic bradyarrhythmia. This study was designed to evaluate the incidence of coexistent CAD in patients with symptomatic bradyarrhythmias and its relationship to conventional coronary risk factors in Chinese people. A total of 113 patients [68 males and 45 females, mean age 70.4±8.2 years old (range 45-86)] were included in our study. The diagnosis was sick sinus syndrome in 69 patients (61%) and atrioventricular block in 44 patients (39%). The incidence of CAD based on coronary angiography was 20%. The nodal-related artery was seldom involved among patients with coexistent CAD and symptomatic bradyarrhythmias (9%), and most patients had significant stenosis over LAD (74%). The

baseline characteristics and presenting symptoms were not different statistically between patients with or without CAD. Hypercholesterolemia (OR 6.6, 95% CI 2.0-22.2, p=0.002) and DM (OR 4.7, 95% CI 1.3-17.2, p=0.020) were the two most significant independent predictors of CAD.

The CAD incidence in the their study was lower compared to those of previous reports. However, they excluded patients with documented myocardial infarction or status post coronary bypass surgery. In addition, evidence of fixed coronary artery stenosis was used for the diagnosis of ischemic heart disease in that study. These factors may contribute to the lower incidence of ischemic heart disease in those present.

The author also discussed as most of their study patients were senile, and most of them had acquired one additional coronary risk factor, they need an aggressive evaluation for ischemic heart disease. However, conventional noninvasive evaluation for coronary artery disease and the left bundle-branch-block ECG pattern after right ventricular pacing are usually not suitable for the diagnosis of CAD in patients with bradyarrhythmia.

On the other hand, routine CAG is costly and low yielding in non-selective patients. However this study revealed that hypercholesterolemia and DM were the two most significant independent coronary risk factors in these patients.

Hence the author suggested that these two factors may be used as clinical markers in selecting patients undergoing further CAG.

Coronary Angiographic Characteristics of Patients With Permanent Artificial Pacemakers

Morris Mosseri, MD. *Circulation.* 1997;96:809-815

The aim of this study was to examine a group of patients with permanent pacemakers who underwent coronary arteriography to determine the extent of coronary atherosclerotic disease that might be responsible for the conduction disturbances.

Forty-three consecutive patients with a permanent pacemaker and 36 matched control patients were investigated. The coronary angiographic study included measurement of diameter and stenosis severity, qualitative assessment of flow, and classification of pathological anatomy, particularly the blood supply to territories supplying the different segments of the conduction system.

Among 43 patients with a permanent pacemaker, 27 had ischemic heart disease (17 after coronary artery bypass graft surgery). The conduction disturbance was infranodal in 28 patients, sinus nodal in 6, AV nodal in 4, and complete AV block of unspecified origin in 5. Patients with permanent pacemakers had a coronary artery pathology compromising blood flow to the septal branches and the right coronary artery (type IV anatomy). This pattern was significantly different from the matched control patients, in whom the most prevalent coronary anatomy was the combination of right coronary artery with distal left anterior descending artery (not involving the septal branches) lesions (P=.007).

Morris Mosseri concluded that patients with coronary artery disease and severe conduction disturbances that require implantation of permanent pacemakers are more likely to have a specific pathological coronary anatomy that combines a compromised blood flow to the septal branches of the left anterior descending artery with right coronary artery lesions. The location of lesions in the coronary tree rather than severe diffuse atherosclerosis appears to be responsible for the conduction disturbances.

AIM OF THE STUDY

The aim of this study was to examine the coronary angiography pattern for patients who were undergoing permanent pacemaker implantation to determine the common pathological and anatomic basis for conduction disturbances.

MATERIALS AND METHODS

Selection of Study group.

This was a prospective study done at the Department of Cardiology, Government General hospital, Chennai. The patients who were referred to our department for the evaluation and management of symptomatic bradyarrhythmias formed the study cohort.

The study population consisted of 36 consecutive patients who had symptoms of bradyarrhythmias due to sick sinus syndrome (SSS) and atrioventricular block (AVB), who underwent permanent pacemaker implantation between January 2004- June 2005 .

None of these patients received ß-blockers, calcium channel blockers or other medications responsible for sinus node dysfunction or conduction disturbances. All patients underwent coronary angiogram prior to or at the time of permanent pacemaker implantation. Patients with valvular heart disease ,congenital heart disease, cardiomyopathy and renal failure (serum creatinine >3.0 mg/*d*l), were excluded.

Inclusion and Exclusion Criteria:

Inclusion Criteria
Patients with symptomatic Bradyarrhythmias
Age > 18
Exclusion Criteria
Hemodyamnically significant Valvular heart disease,
Congenital heart disease,
Cardiomyopathy
Renal failure((serum creatinine >3.0 mg/*d*l).

Clinical Data

The clinical parameters, demographic data and echocardiographic findings were collected for all the patients. A detailed history of symptoms were recorded. Low cardiac output was defined as upright dizziness, fainting, lethargy, or decreased workload. The presentations of pulmonary congestion included dyspnoea on exertion, paroxysmal nocturnal dyspnoea, or frank pulmonary edema.

Presentation of angina included chest tightness with typical or atypical characteristics. Syncope was defined as transient loss of consciousness with spontaneous recovery and without neurological sequelae. Subjective sensation of an irregular heart rhythm was considered to be a symptom of palpitation.

Central nervous system (CNS) symptoms included transient ischemic attack (TIA) or cerebral infarction with evidence of focal neurological sequelae. Details of underlying cardiac disease, blood pressure, and fasting blood glucose and serum cholesterol levels were measured. The **c**onventional coronary risk factors including

malesex, age, diabetes mellitus (DM), hypertension, hypercholesterolemia, smoking obesity,family history of CAD and physical inactivity were recorded.

The presence of DM and hypertension were determined by history and from old medical records. A fasting total serum cholesterol level above 200 mg/*dl* on two sequential blood tests was considered as hypercholesterolemia. Our definition of smoking risk factor requires at least 10 years exposure to tobacco with at least 10 cigarettes per day.

Mean age of the patients was 51.6 years (range 24 to 71), 15 were men (42%) and 21(58%) were females. Thirteen patients (36%) had angina ,among them eight (61.5%) patients had typical angina, 5 patients (38.5%) had atypical angina. Relevant demographic data of all patients is displayed in table 1

Definition of SSS and Complete AV Block

The diagnosis of bradyarrhythmias, which included sick sinus syndrome (SSS) and atrioventricular conduction disturbances (AVD), should be established by 12-lead surface ECG or 24-hour ambulatory ECG study.

Inappropriate sinus bradycardia

Sinus bradycardia (sinus rate less than 60 beats/min) considered inappropriate when it is persistent and does not increase appropriately with exercise.

Sinus Arrest

The terms sinus arrest and sinus pause is defined as pauses greater than 3 seconds, the pause is not an exact multiple of the preceding PP interval

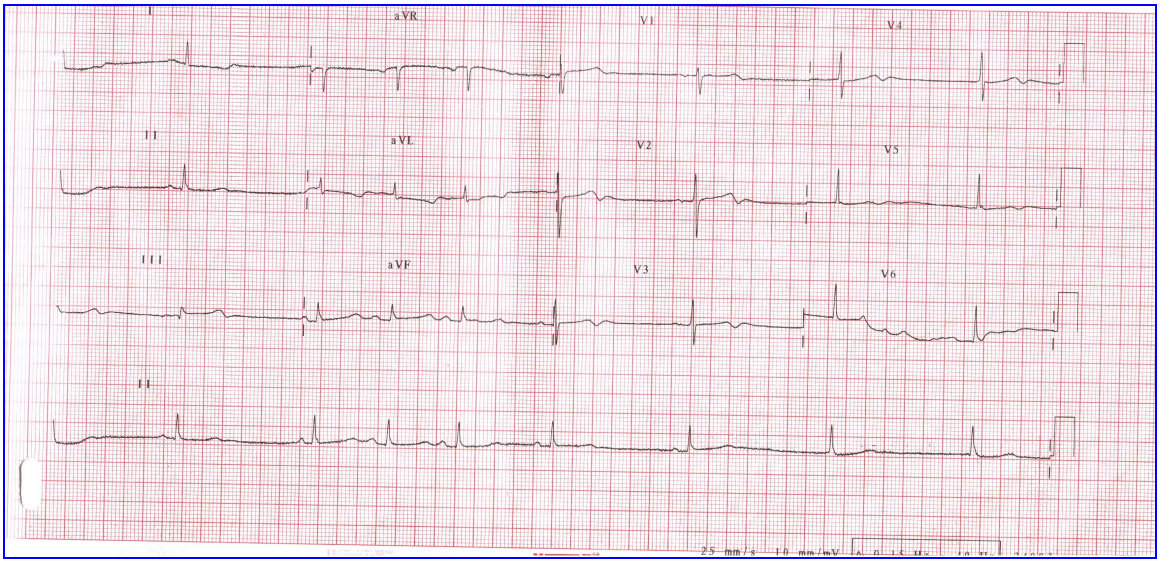
Sinoatrial Exit Block

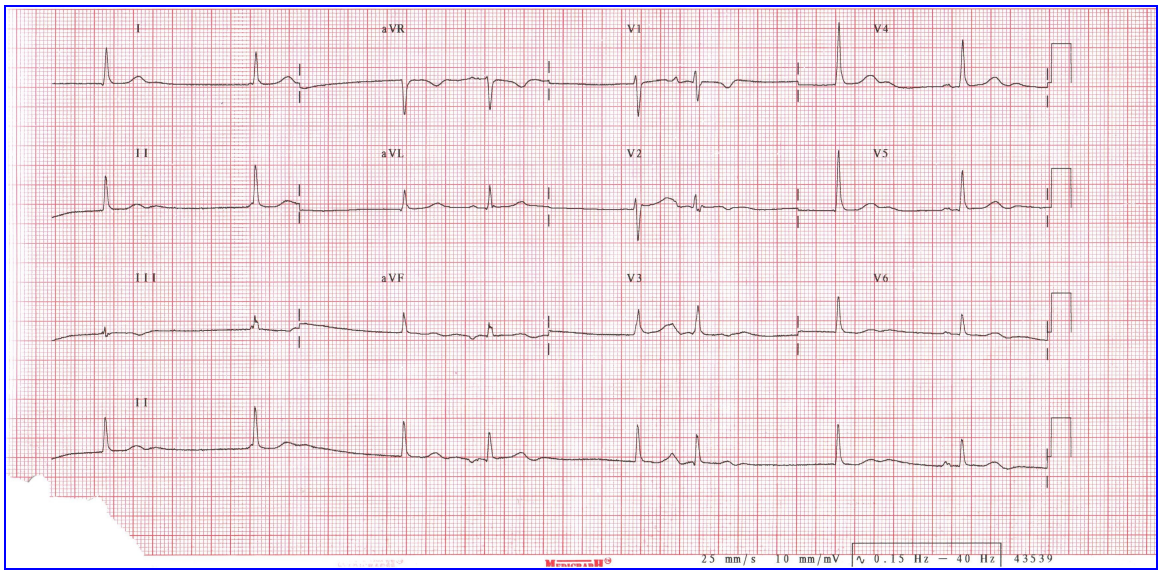
Sinoatrial exit block is characterized by a period of asystole without the expected P wave seen. [pause is an exact multiple of the baseline PP interval]

Tachycardia-Bradycardia Syndrome

Tachycardia-bradycardia syndrome refers to the presence of intermittent sinus or junctional bradycardia alternating with atrial tachycardia (usually paroxysmal atrial fibrillation) in the same patient

Sinus pause /sinus arrest in a patient with SSS

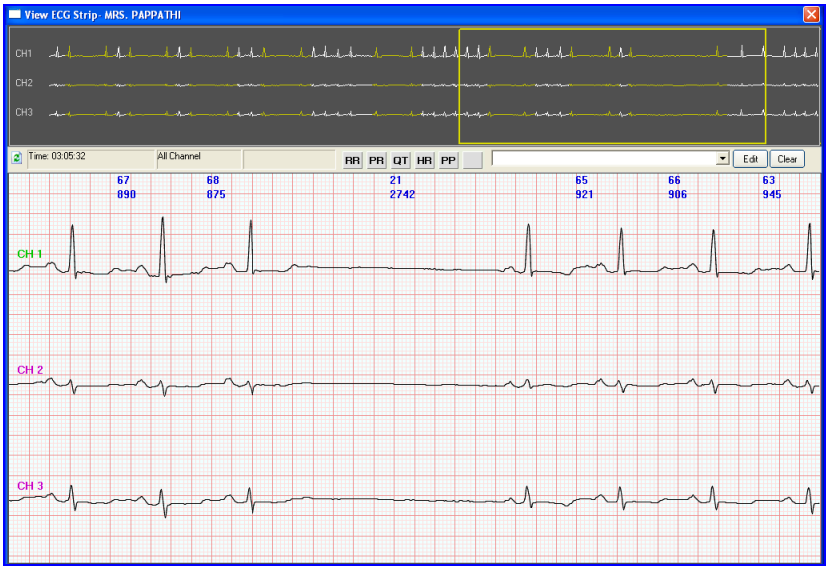


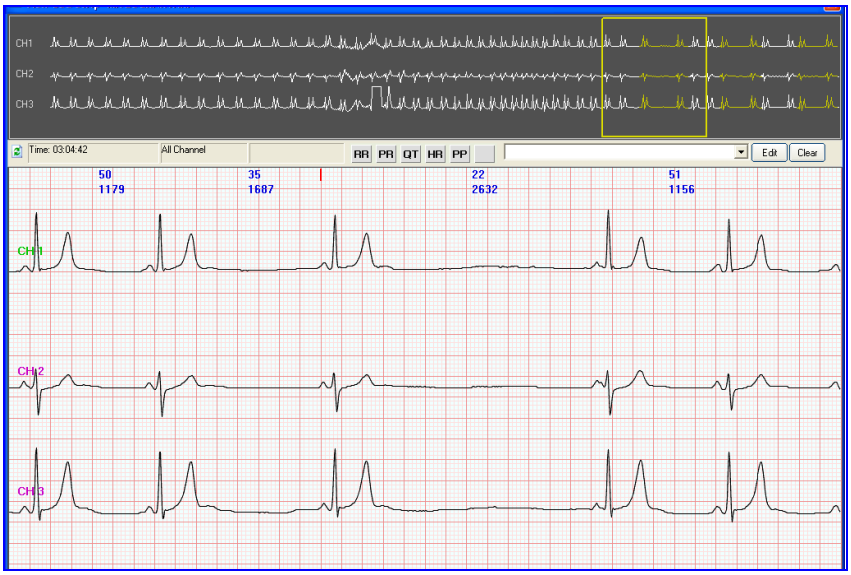


Sick Sinus Syndrome

Holter study : 1

Sinus Pause / Sinus Arrest





Chronic Atrial Fibrillation

The presence of chronic atrial fibrillation in a patient with a slow ventricular response not secondary to drug therapy is a sign of sinus nodal dysfunction.

AV Block

First-degree AV block

Defined as PR interval >0.20 seconds in adults with symptoms.

Type II, or Mobitz II, second-degree AV block

Characterized on the surface electrocardiogram by a constant PR interval,

followed by sudden failure of conduction P wave to the ventricles. The PP intervals should remain constant, and the pause including the blocked P wave equals two PP intervals.

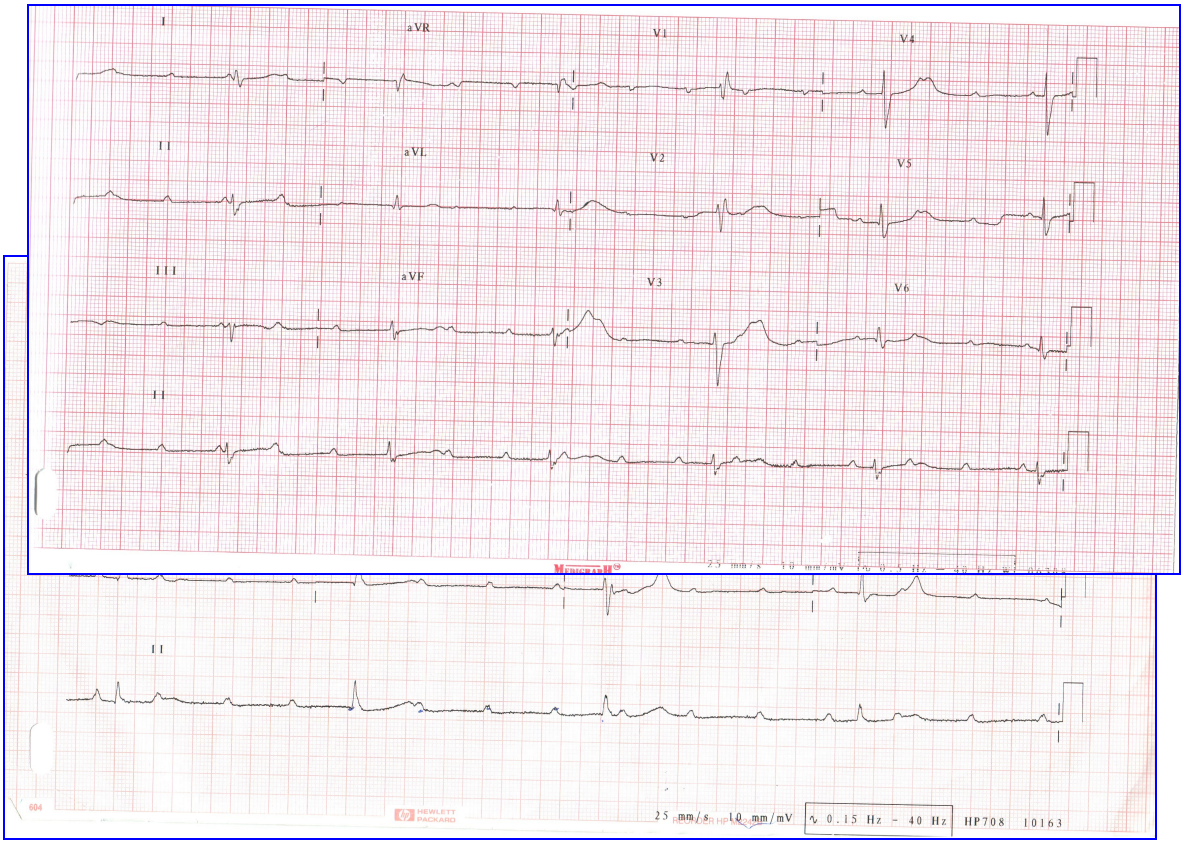
2:1 Atrioventricular Block

Characterised by failure of conduction of alternate P waves

High-Degree AV Block

Nonconduction of two or more consecutive P waves when AV synchrony is otherwise maintained is sometimes termed high-degree AV block.

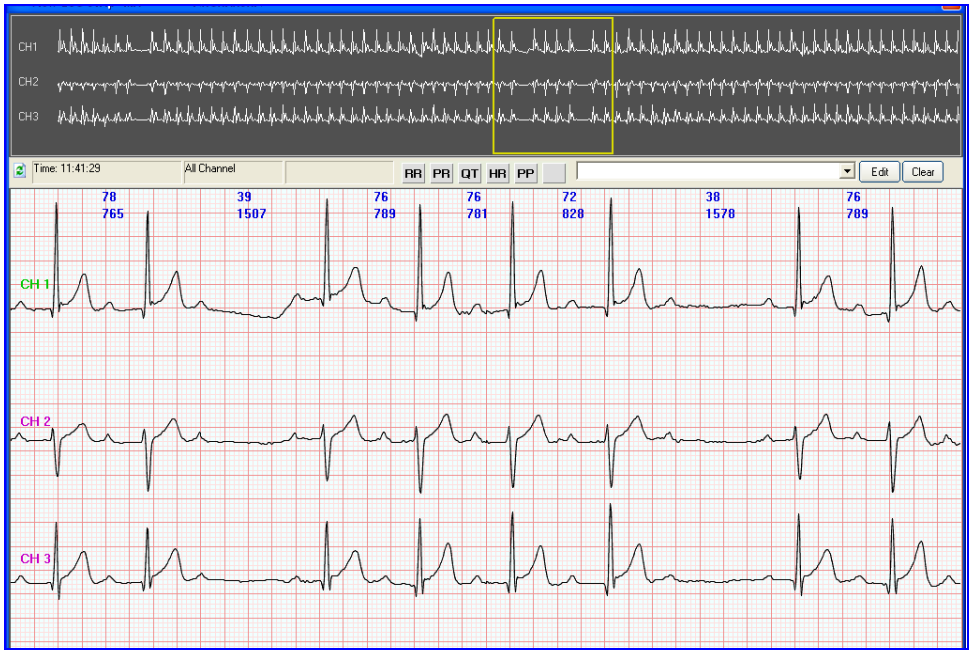
III degree AV block



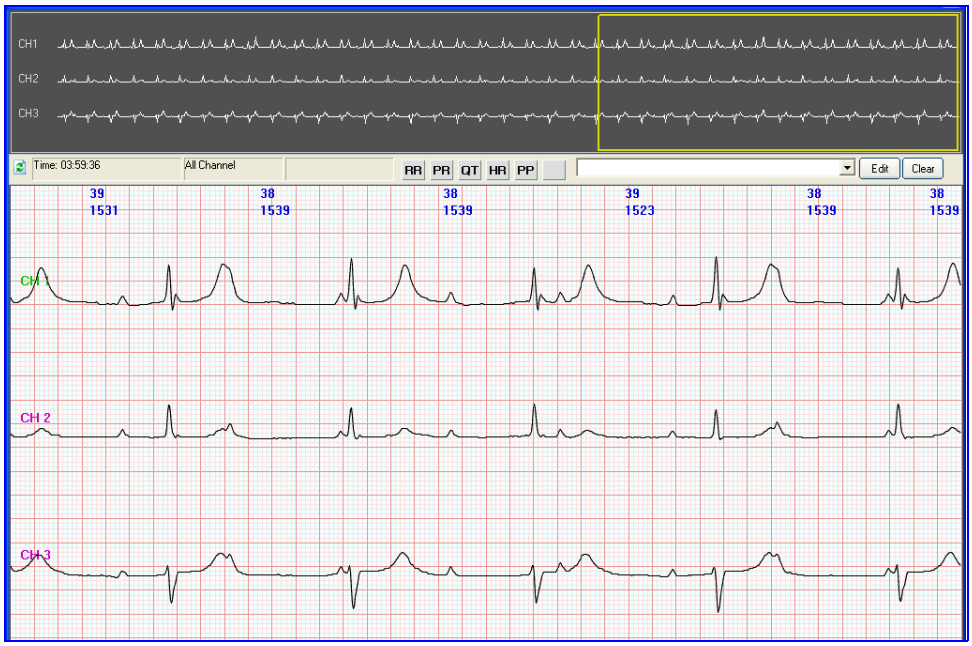
Atrioventricular Block

Holter Study

Second-Degree Atrioventricular Block



Third-Degree Atrioventricular Block



Third-degree, or complete, AV block

Characterized on the surface electrogram as completely dissociated P waves and QRS complexes, each firing at their own pacemaker rate.

Interpretation of coronary angiography:

Coronary angiography was performed during the same session as pacemaker implantation. A total of 3-4 views for left coronary artery and two views for right coronary artery were done. The coronary angiography was reviewed by two independent experienced cardiologists unaware of the patient's general data. The Coronary angiographic study included measurement of diameter and stenosis severity, qualitative assessment of flow particularly the blood supply to territories that supply conduction system(proximal LAD , Right coronary artery and Left Circumflex coronary artery) were documented.

Luminal stenosis was calculated as the percentage of diameter reduction in diseased segment compared to the proximal disease free reference segment. More than 50% stenosis in either one of three major coronary arteries including the left anterior descending artery (LAD), left circumflex artery (LCX), and right coronary artery (RCA) or their first-order branches was considered as significant for CAD. The sinus nodal artery (SNA) and atrio-ventricular nodal artery (AVNA) were identified. Significant flow compromise was defined as more than 50% stenosis in the nodal

artery or its feeding artery proximal to the origin.

The qualitative assessment of antegrade flow and retrograde flow to branches supplying the conduction system were graded qualitatively in each patient as good, moderate and poor.

Results

A total of 36 patients [15 males and 21 females, mean age of 51.6 years old (range 24-71) were included in our study. The diagnosis of sick sinus syndrome in 10 patients (28%) and atrioventricular block in 26 patients (72%).

Table 1. Demographic Data

	Normal CAG (No: 25)	Abnormal CAG (No:11)	p- value
No. of patients	25(69%)	11(31%)	
Age,years (mean)	24-71 (41.8)	50-70(58.7)	
Male	9(36%)	6 (55%)	<0.05
Female	16(64%)	5 (45%)	<0.05
Sick sinus syndrome	8(32%)	2 (18%)	NS
Atrio-ventricular block	17(68%)	9 (82%)	NS

Table 2. Presenting Symptoms

	Normal CAG (No: 25)	Abnormal CAG (No:11)	p- value
Angina	7 (28%)	6(54%)	<0.05
Syncope	18(72%)	4(36%)	<0.05
Palpitation	2 (8%)	1 (9%)	NS
Low cardiac output	1 (4%)	3 (27%)	<0.05
Cardiac congestion	2 (8%)	2 (18%)	NS
CNS symptoms	2 (8%)	1 (9%)	NS

There were no significant differences in baseline characteristics and presenting symptoms between patients with or without CAD. The three most frequent complaints for patients with abnormal CAG were angina (54%) syncope (36%) and symptoms of low cardiac output (27%). For patients with normal CAG, syncope was the most common symptom and observed in 72% of patients. Angina and low cardiac output were more common in patients with CAD group.

Table 3. Distribution of Conventional Coronary Risk Factors

	Normal CAG (No: 25)	**Abnormal CAG** (No:11)	**p- value**
Age- years Mean	42	58.7	
Male sex	9(36%)	6(55%)	NS
Hypercholesterolemia	2 (8%)	4 (36%)	NS
Hypertension	6 (24%)	6 (55%)	NS
Diabetes mellitus	4(16%)	4 (36%)	NS
Smoking	8 (32%)	4 (36%)	NS
Obesity	6(24%)	3(27%)	NS
Physical inactivity	6(24%)	2(18%)	NS
Family H/O early onset CAD	4(16%)	2(18%)	NS

Regarding coronary risk factors, patients with abnormal CAG had significantly higher percentages of male sex (55% vs 36%) , hypertension (55% vs 24%), hypercholesterolemia (36% vs 8%) and Diabetes mellitus (36% vs 16%); Table-3.

Multivariate logistic regression analysis of conventional coronary risk factors also revealed male sex, hypertension, hypercholesterolemia and Diabetes mellitus were the significant predictors of CAD (Table-4). The odds-ratio for male sex is 2.13, hypertension - 3.8, hypercholesterolemia –6.57 and for diabetes mellitus is 3.0.

Table 4. . Multivariate analysis of Coronary Risk Factors

Risk Factors	OR
Male sex	2.13
Hypercholesterolemia	6.57
Hypertension	3.8
Diabetes mellitus	3.0
Smoking	1.21
Obesity	1.19
Physical inactivity	0.7
Family H/O early onset CAD	1.17

In our patients with symptomatic bradyarrhythmias requiring permanent cardiac pacing, the incidence of CAD was 31% as determined by coronary angiography (CAG). For patients with sick sinus syndrome, coronary artery disease incidence was 20% and in atrioventricular block was 35%. The sinus nodal-related artery was seldom involved among patients with coexistent CAD and symptomatic bradyarrhythmias (9%), and most patients (90%) had significant stenosis over LAD.

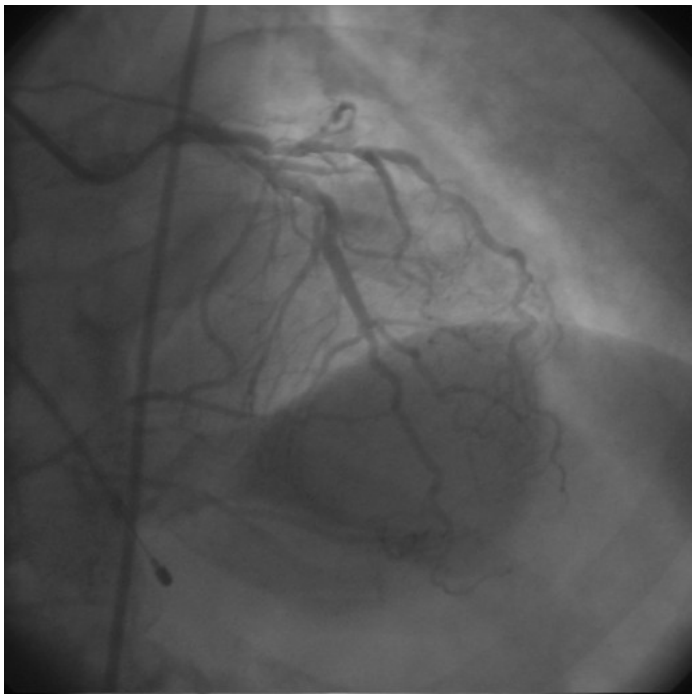
Table 5. Coronary arterial profile	
Right Dominance	80.6% (29 / 36)
Left Dominance	13.8% (5 / 36)
Codminance	5.6 % (2 /36)
SA nodal artery	
RCA	61% (22/36)
LCx	49% (14 /36)
AV nodal artery	
RCA	83.4%(30 / 36)
LCx	8.3% (3/36)
Not visualised	8.3% (3 /36)

We further divided patients with CAD into 2 subgroups based on their diagnoses of bradyarrhythmia. The findings of their coronary angiographies are presented in Table -5 and 6. Both subgroups had similar distributions with respect to the coronary arteries involved. Most patients had significant stenosis over LAD (>90% patients). The sinus nodal-artery insufficiency was quite infrequent among our patients with SSS (5.5%). One patient in the both SSS group and AVB had sinus nodal artery involvement. AV nodal artery involvement was 13%, all of which occurred in the AV block group.

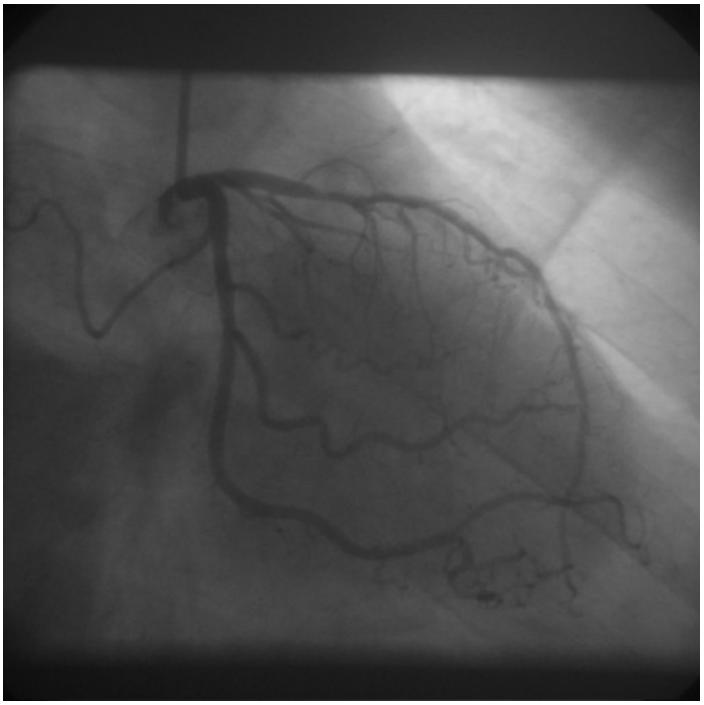
Table 6. Distribution of Involved Coronary Arteries in Different Diagnosis

	SSS No: 2/10	**AV block No: 9/26**
LAD	2	8
LCX	0	1
RCA	2	5
SN artery	1	1
AVN artery	0	5

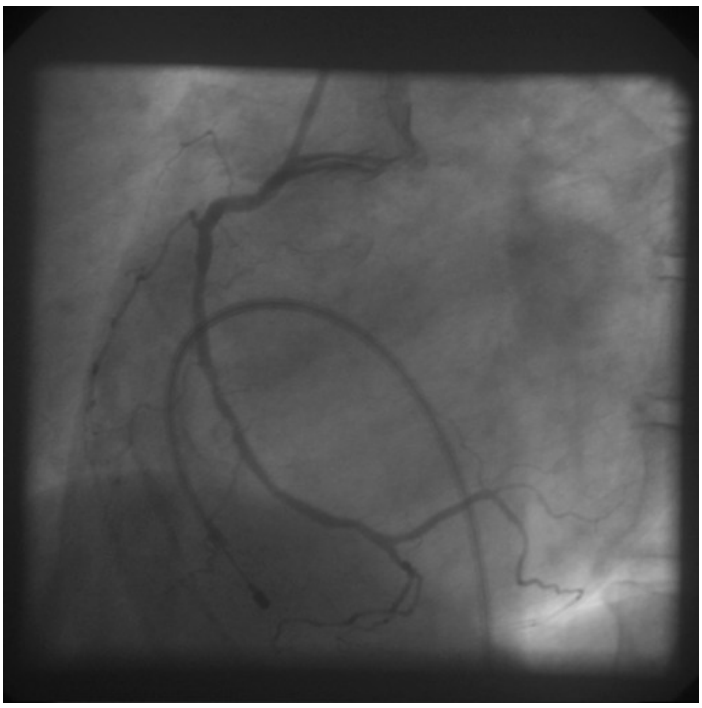
LAD and LCX Lesion, Collaterals to RCA in case of nodal AV block



LAD Disease in a patient with infranodal AV block



Mid RCA / AV nodal artery involvement in AV block patient; LAO



Mid RCA / AV nodal artery disease in a patient with AV block; RAO

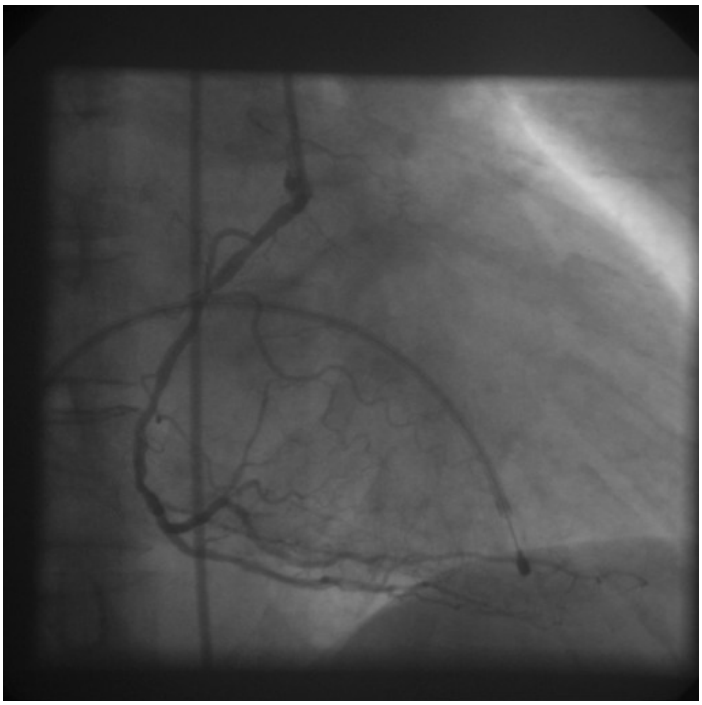


Table 7. Flow Quality in Septal Branches, SAN and AVN arteries

	Septal Branches of LAD	SAN artery	AVN artery
Moderate to Good	5	9	4
Poor	6	2	7
Total	11	11	11

Table 8. Clinical Diagnosis, Location of Conduction Disturbance and Involved coronary arteries

S.NO	Clinical Diagnosis	Location of Conduction Disturbance	Coronary arteries involved
1	SSS	SA node	LAD, RCA
2	SSS	SA node	LAD, RCA
3	AVB	AV node	LAD
4	AVB	AV node	LAD, RCA
5	AVB	AV node	LCX
6	AVB	Infranodal	LAD, RCA
7	AVB	Infranodal	LAD, RCA
8	AVB	Infranodal	LAD
9	AVB	Infranodal	LAD
10	AVB	Infranodal	LAD, RCA
11	AVB	Infranodal	LAD, RCA

Statistical analysis.

The patients were divided into two groups based on their coronary angiographic findings. All parametric data are expressed as the mean (± SD of the measured variables. Intergroup differences in means were checked by two-sided unpaired Student's *t*-test. The categorical variables were measured by the chi-square test with or without Yate's correction as needed. Multivariate logistic regression analysis was performed to determine independent predictors of CAD including sex, age (dichotomized at 70 years old), DM, hypertension, hypercholesterolemia, and smoking. The odds ratios and 95% confidence intervals of independent factors were calculated. A *p* value of less than 0.05 was considered significant.

Discussion

There are very few studies concerning the incidence of CAD in patients receiving permanent pacemaker implantation for symptomatic bradyarrhythmia. Our study results have revealed that 31 % of patients with symptomatic bradyarrhythmias had coexistent CAD.

Rubenstein[1] *et al* found that 20 of their 56 patients with sick sinus syndrome had coronary artery disease based on either historical or electrocardiographic evidence;

Shaw[2], *et al* reported that 12 of their 25 patients with sick sinus syndrome had abnormal postmortem CAG.

Hsueh[3], et al reported a prevalence of 20% CAD in their study

The CAD incidence in the present study was similiar to those of previous reports.

Nearly half of our study patients complained of chest tightness with either typical or atypical characteristics. The patients were mean of more than 58 years old and most of them acquired one additional coronary risk factor. Furthermore, considering the major prognostic role of CAD in patients undergoing permanent cardiac pacing, these patients dictate an aggressive evaluation for ischemic heart disease.

However, patients with bradyarrhythmia are usually not suitable for conventional noninvasive evaluation for coronary artery disease, and the left bundle-branch-block ECG pattern after right ventricular pacing, is invalid for the diagnosis of CAD. On the other hand, routine CAG is costly and low yielding in non-selective patients. Our study revealed that male sex, hypertension, hypercholesterolemia and DM were more prevalent coronary risk factors in these patients. These factors may be used as clinical markers in selecting patients undergoing further CAG. The distribution of diseased vessels in our

patients was not different between the two different bradyarrhythmia diagnoses. LAD was the most frequent vessel involved. Sinus nodal artery insufficiency was noted in one patient among 10 patients diagnosed with sick sinus syndrome. Atrioventricular nodal artery insufficiency was noted in five patients among 26 patients with the diagnosis of high degree atrioventricular block.

Jordan.[9], *et al* found significant sinoatrial conduction time (SACT) prolongation in patients with sinus nodal artery insufficiency and suggested a pathogenetic role of CAD in the development of sinus nodal dysfunction. However, Shaw, *et al* reported only 4 of 25 patients with chronic sinoatrial disorder had angiographic evidence of more then 75% stenosis obstructing the sinus nodal artery. Our data disclosed that a sinus nodal related artery was seldom involved in patients with symptomatic bradyarrhythmia and coexistent CAD.

Contrary to earlier studies, AV nodal artery involvement was significantly higher in our patients with AV block. This could be due to inclusion of patients with documented myocardial infarction in the past.

Although our study population was small and confined to south Indian population, the results suggest that the prevalence of CAD is significant in patients with symptomatic bradyarrhythmia.

Study limitations.

Our cohort of patients with permanent pacemakers who also underwent coronary angiography is biased, has both patients with or without ischemic heart disease, and does not represent the general population of patients with a permanent pacemaker.

Nevertheless, the comparison of the study group with a matched control group is valid and yields meaningful results.

Coronary angiography is not indicated routinely in patients with permanent pacemakers but there is no other way to study the pathological anatomy of these patients.

Conclusions

- In conclusion, coexistent CAD was noted in a significant number (31%) of our patients receiving permanent pacemaker implantation for symptomatic bradyarrhythmia.

- Among the conventional coronary risk factors, male sex, hypertension, DM and hypercholesterolemia were the significant predictors and may act as important markers for the judgment of further coronary artery evaluation.

- The AV nodal artery was commonly involved among patients with bradyarrhythmia due to AV block and coexistent CAD.

Bibilography

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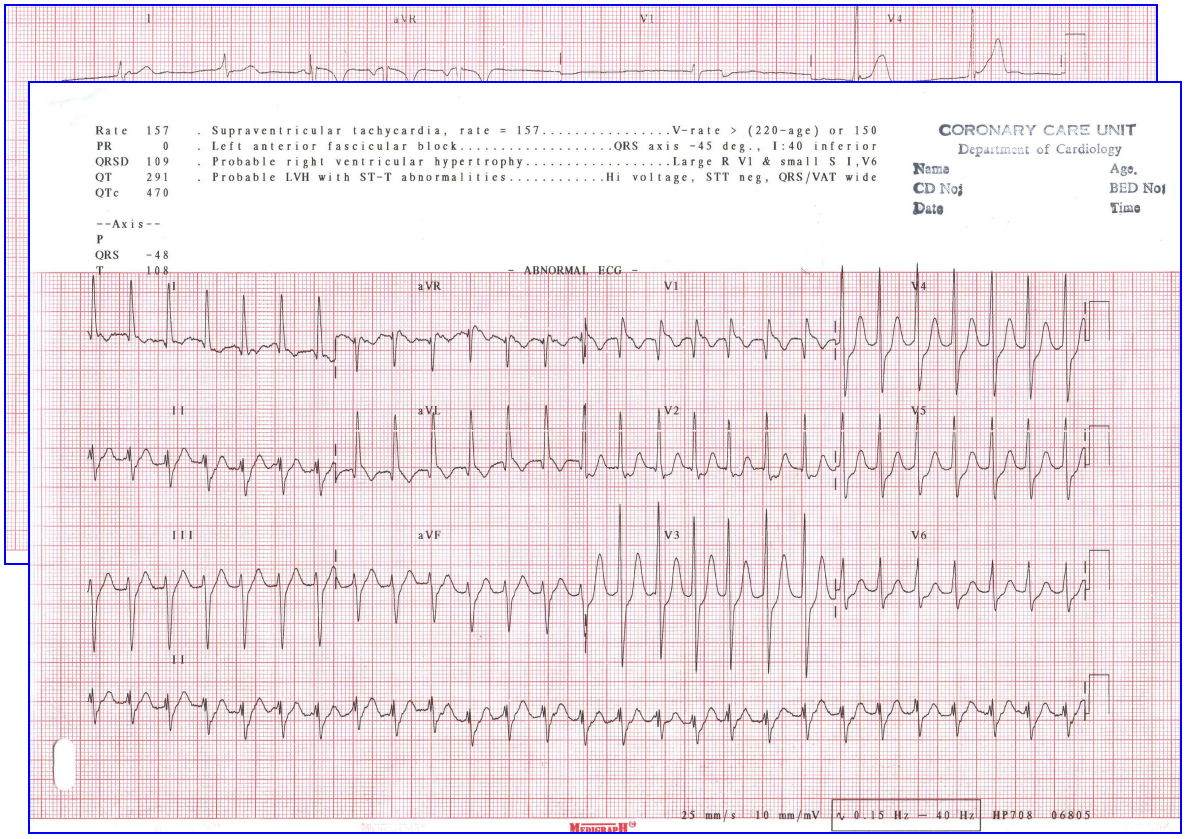
Appendix

Sick Sinus Syndrome ; Tachycardia-bradycardia syndrome



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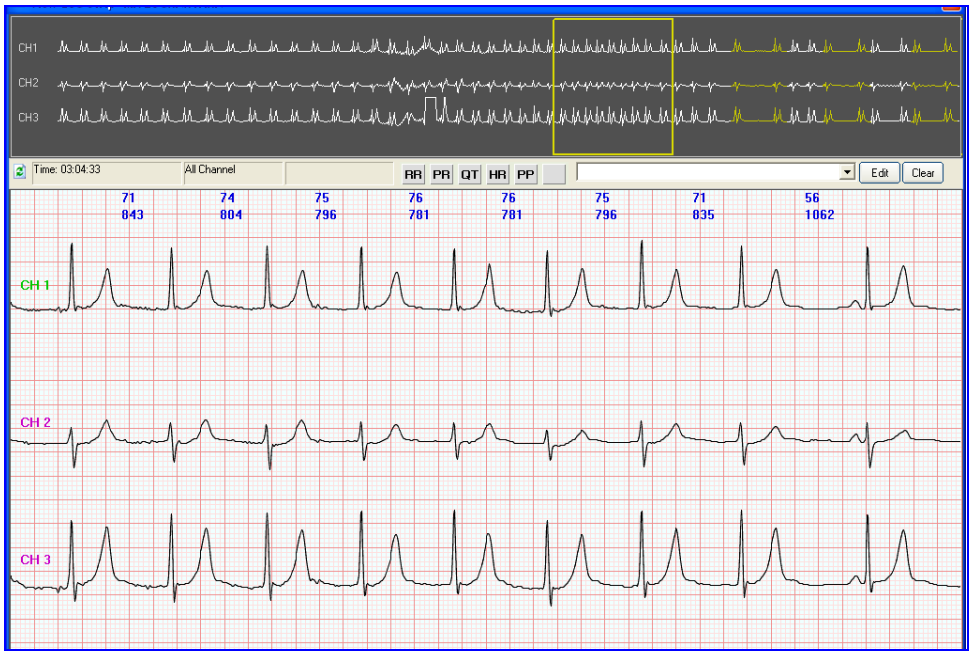
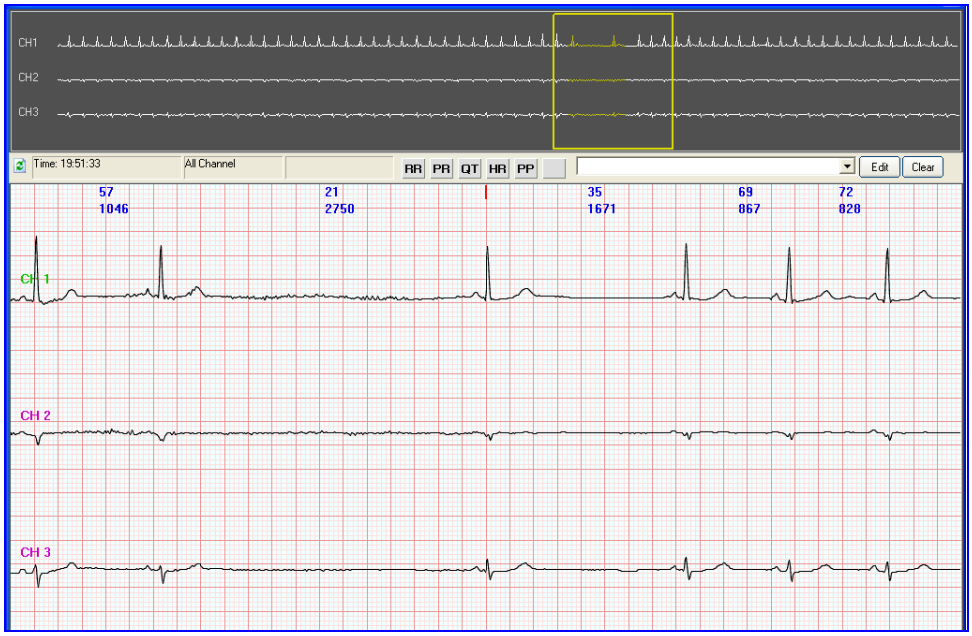
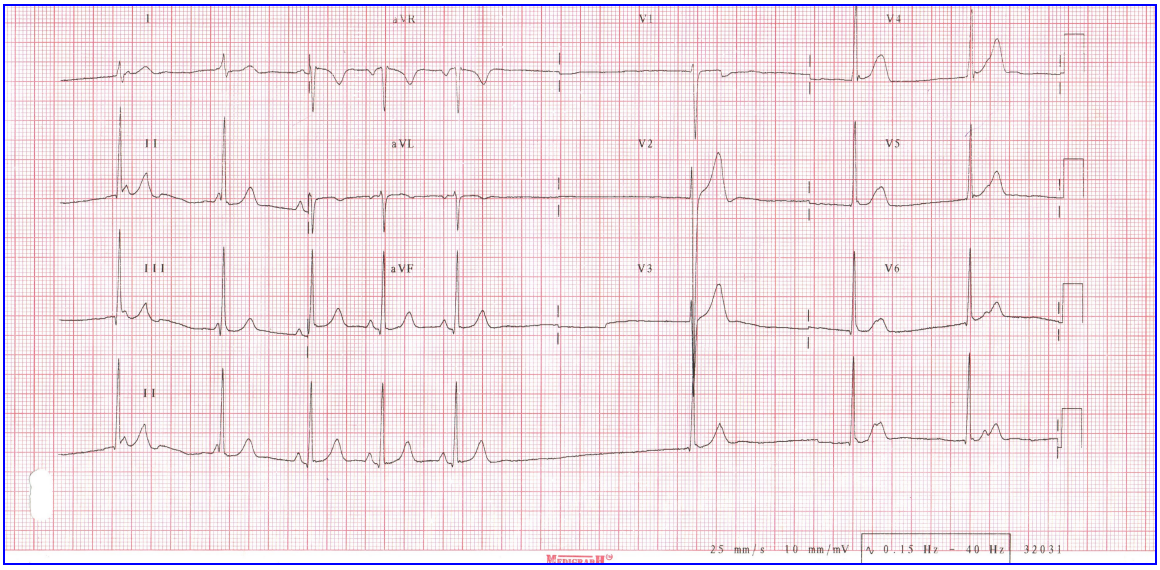
Tachycardia-Bradycardia Syndrome

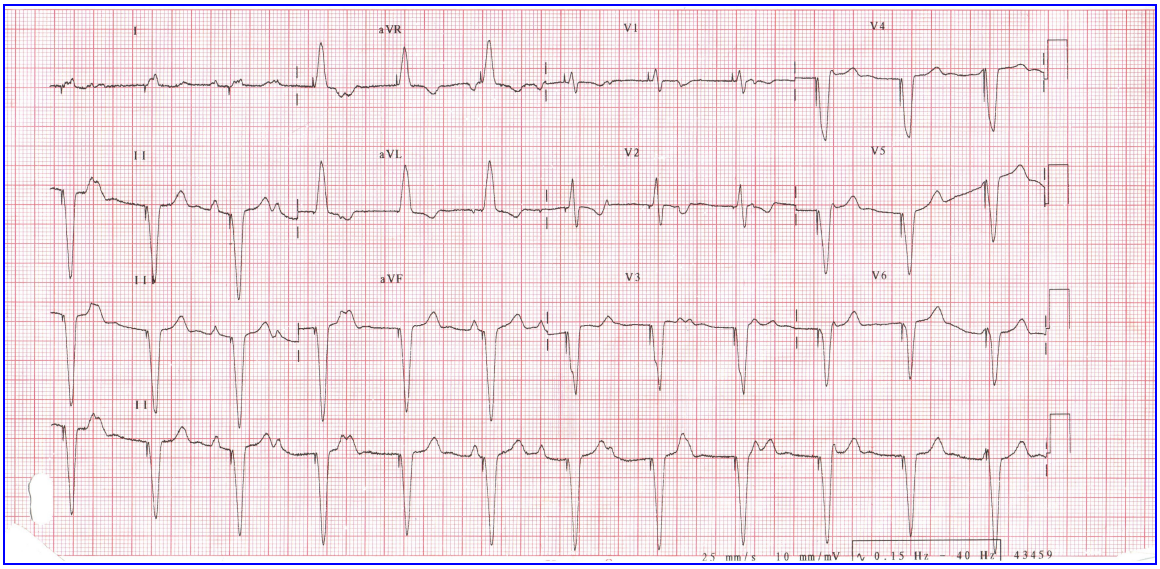




Sinus arrest in a patient with SSS

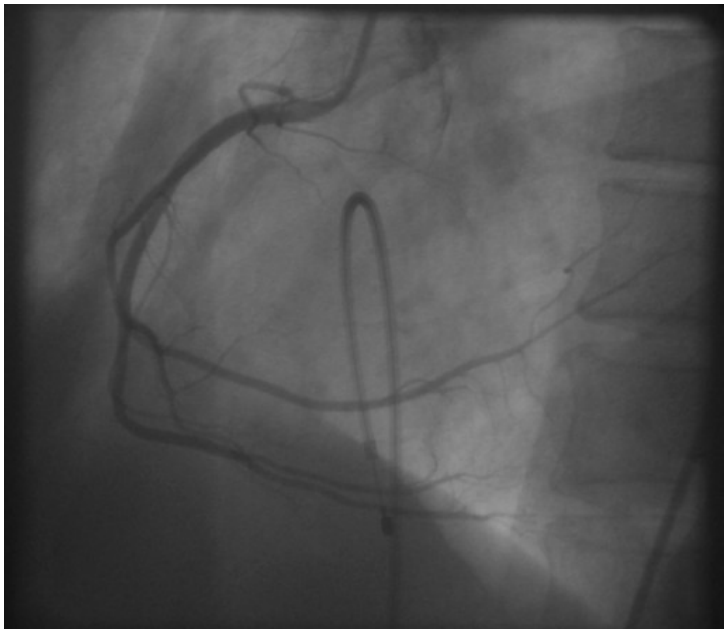


Post PPI; RV paced rhythm in a patient with permanent pacemaker



Coronary angiogram

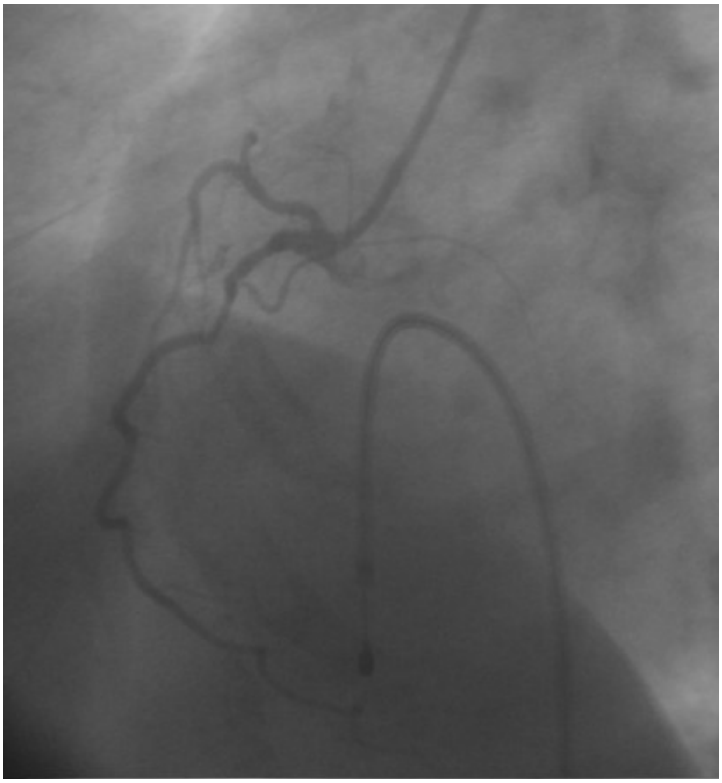
Normal RCA angiogram

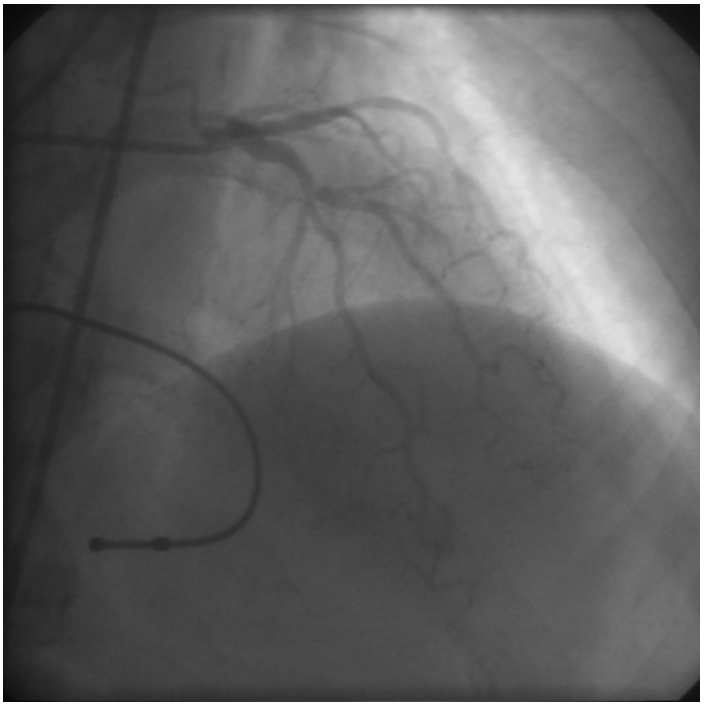


Sick sinus syndrome

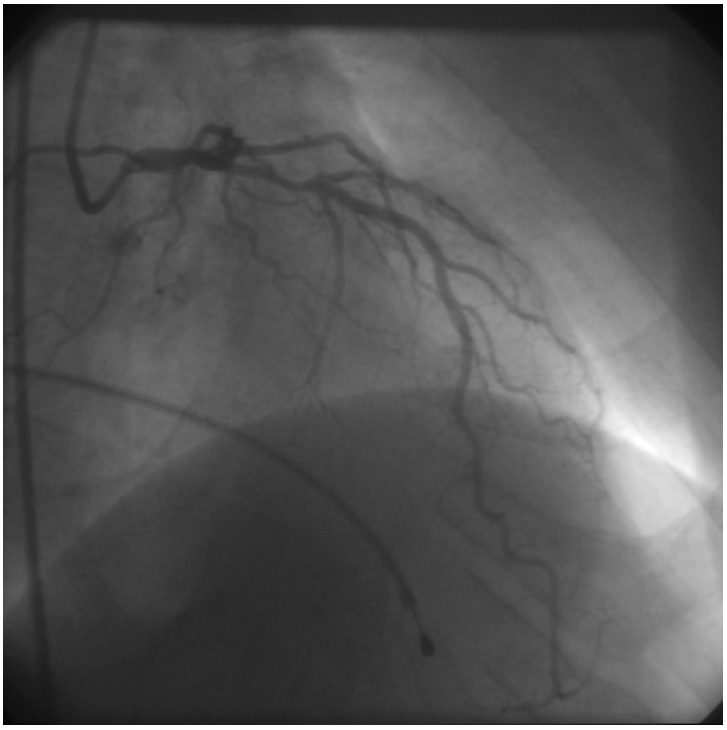
- **100% total occlusion proximal RCA .**
- **SA node artery arising astride the lesion.**

Proximal LAD diseae in a patient with infranodal disease

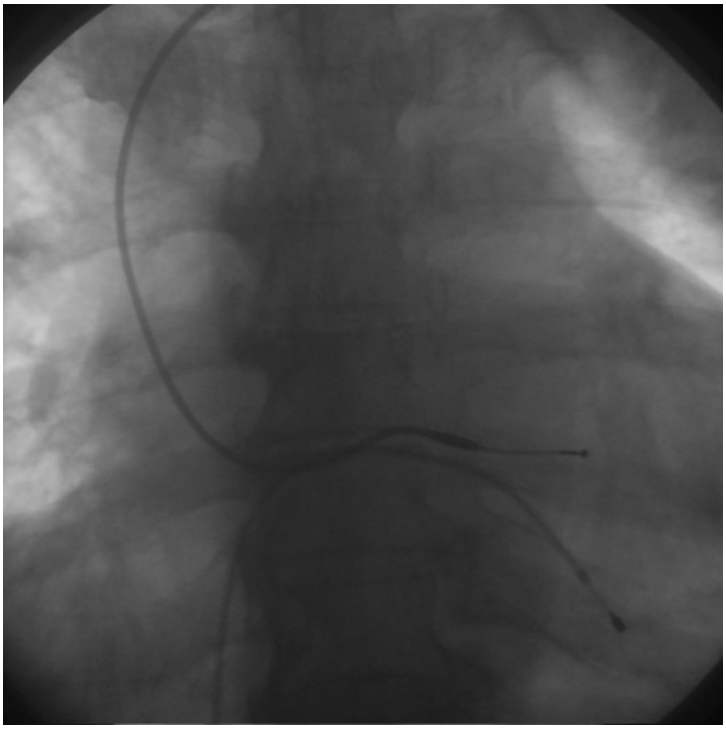


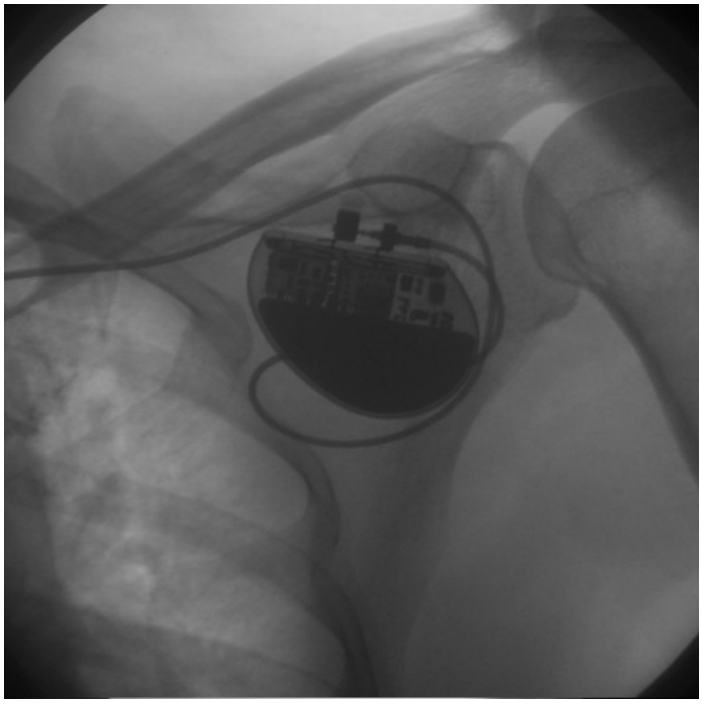


LAD disease in a patient with AV block



Permanent pacemaker implantation in a patient with SSS





LIST OF ABBREVIATIONS

AVN = artery to AV node

AVB = atrioventricular block

CAD= coronary artery disease

LAD = left anterior descending coronary artery

LAO = left anterior oblique

LCx = left circumflex coronary artery

PDA = posterior descending artery

PL = posterolateral

PTCA = percutaneous transluminal coronary angioplasty

RAO = right anterior oblique

RCA = right coronary artery

SA = sinoatrial

SAN = artery to sinoatrial node

SSS = Sick Sinus Syndrome